THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—31ST YEAR. SYDNEY, SATURDAY, APRIL 8, 1944. No. 15.

Table of Contents.

ORIGINAL ARTICLES— Page.
The Tongue in Medical Diagnosis, by Douglas Anderson, M.D. .. 309
A Plea for the Standardization of the Lepromin Test, by John W. Fielding and Robert G. Cochrane .. 313
Treatment during Convalescence after Head Injury, by J. Estcourt Hughes, M.S., F.R.A.C.S. .. 316
A Brief Report on the Value of the Selective Medium of Wilson and Blair for the Isolation of Dysentery Bacilli, by T. S. Gregory .. 319
Meningitis due to Hæmophilus Influenzæ: Review of Treatment, by A. G. Nicholson, M.B., B.S. .. 320

REVIEWS—
Unusual Bone Diseases .. 322

LEADING ARTICLES—
The Outbreak of Enteric Fever at Moorabbin .. 323

CURRENT COMMENT—
Liver Function Tests .. 324
Confinement to Bed after Surgical Operation .. 324
Wheat Breakfast Foods .. 325

ABSTRACTS FROM MEDICAL LITERATURE—
Pædiatrics .. 326
Orthopædic Surgery .. 327

MEDICAL SOCIETIES— Page.
Melbourne Pædiatric Society .. 328

NAVAL, MILITARY AND AIR FORCE—
Clinical Meeting at an Australian General Hospital 329
Appointments .. 330

CORRESPONDENCE—
Housekeepers' Emergency Service .. 330
Health and the Gold Standard .. 330
Acute Otitis Media—Paracentesis—Sulphonamide Drugs .. 331
Industrial Medicine .. 331

OBITUARY—
Horace Frederick Hayes .. 332
Roy William Chambers .. 332
Hercules Bradshaw Moorhead .. 332

AUSTRALIAN MEDICAL BOARD PROCEEDINGS—
Tasmania .. 332

MEDICAL APPOINTMENTS .. 332

DIARY FOR THE MONTH .. 332

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 332

EDITORIAL NOTICES .. 332

THE TONGUE IN MEDICAL DIAGNOSIS.[1]

By DOUGLAS ANDERSON, M.D.,

Honorary Assistant Physician, the Royal North Shore Hospital of Sydney.

THE doctor, in former times, whether at the bedside or in the consulting room, never omitted to inspect the patient's tongue, just as he never omitted to count the pulse by his great gold watch. I do not believe that he looked at the tongue with an unseeing eye, and I do not doubt that what he saw helped him materially in his summing-up of the patient's state of health. The tongue, it was said, in its communications to our auditory sense, is often a sorry deceiver, but to our visual sense it always tells the truth. Yet at the present day the tongue is almost entirely neglected by the clinician except in special cases. No mention of the tongue in medical diagnosis is to be found in some of the leading text-books of medical practice. Most of the text-books of clinical methods describe the methods of examining the tongue, but some take the trouble to discount the value of the information to be gained from it. It is interesting to try to discover the reasons for this change of outlook, which has coincided with the growth of scientific medicine.

I believe that the older physicians interpreted what they saw in the tongue in the most elementary kind of way; they regarded a coat on the tongue in much the same way as they might have regarded a pale countenance or an anxious expression or a shifty eye, as an element which they could add to other elements to make, not a diagnosis, but an appreciation of the state of health. It was natural that the inquiring mind should try to translate these bedside impressions into precise terms, to explain just exactly what the coated tongue meant. And I think they asked the tongue to explain too much. For a long time a mysterious sympathy was supposed to exist between the tongue and the alimentary canal. It was very plausible for our medical forefathers to say that the coated tongue was a sure sign that the stomach was out of order. The inevitable sequel was a grey powder or a blue pill, and the doctor was able to display himself as a man of prompt discernment and ready execution. And it was not only our forefathers who argued and proceeded in this way, but our fathers themselves, and that down to a very recent day. A more critical generation has since arisen; it has sought out the truth by way of experiment; it has examined such beliefs as that "the tongue is the mirror of the intestine" and such dicta as "raw-red tongue, raw-red gut", and found them unfounded; but the evidence adduced has been conflicting, appearing at one time to corroborate, at another to contradict. Now nobody is certain what is the exact meaning of the coated tongue (except the patent medicine proprietors), and we of the present generation, baffled by the mass of knowledge, are apt to leave the coated tongue out of our calculations altogether. We can do this with a conscience the clearer because there is much new knowledge of the state of the tongue in metabolic disease that is exact and helpful in diagnosis and treatment. Also, there have been so many of these useful observations on the state of the tongue in general diseases, that most modern writers on the present subject find ample scope in expounding and classifying these, without giving more than a passing reference to the cause of those abnormalities which occur the most frequently and are the most baffling and the most important in everyday medical practice—the dry tongue, the coated tongue, the sore tongue and the tremulous tongue. It is of these abnormalities that I shall treat particularly in this paper.

[1] Read at a meeting of the Section of Medicine of the New South Wales Branch of the British Medical Association on September 16, 1943.

The Dry Tongue.

Persistent and considerable dryness of the tongue implies as a rule failure of the salivary secretion. The lack of saliva is declared by the loss of the power of spitting; also lemon juice on the tongue fails to produce a flow of saliva from the parotid duct. Boerhaave, the celebrated Dutch physician who flourished at the beginning of the eighteenth century, said: "The saliva continually flows into the mouth of a person in health and nothing is a surer sign to a physician of disease in a patient than his having a dry mouth." I think these words are true today. What is the cause of the diminished flow of saliva? This is a question which so far as I can discover no one has ever attempted to answer by considering the possible ways in which the physiological processes concerned in salivation may be deranged.

During the act of salivation several physical and vital processes take place in the salivary glands. Exactly what they are is not fully understood. It is thought that first of all the blood flow through the gland is greatly increased, partly by nervous and partly by local chemical influences on the blood vessels in it, so that an abundant source of fluid is supplied to the gland; on account of the rise in capillary blood pressure the transfer of some of this fluid to the tissues is greatly facilitated. Secondly, in response to the activity of the secretomotor nerves, large non-osmotic molecules in the granules of the gland cells are broken down into numerous small molecules, which are osmotically active and attract from the blood and retain large quantities of fluid. Thirdly, the granules of the gland together with the water which they have attracted are discharged at the periphery of the gland cells into the lumen as saliva. From such a conception of the physiology of salivation several possible ways suggest themselves whereby the flow of saliva could be reduced.

First of all, if the parasympathetic nervous tone is diminished or the sympathetic tone increased or both, the nervous mechanism of salivation is disturbed. This may happen in states of nervous tension, and the result (as many a candidate for *viva voce* examination knows) is a diminution in the secretion of watery saliva and the production of small quantities of thick mucinous saliva by the submaxillary glands. Dryness of the tongue from nervous causes is usually transient and is unimportant. Secondly, in a state of anhydræmia the viscosity of the blood would be increased and the effect of an increased capillary bed somewhat offset, so that the effective circulation would be reduced; also the water available for solution of the dissolved contents of the saliva would be less and the formation of saliva from its precursor substances retarded; also possibly, if the circulation were reduced sufficiently, a mild degree of stagnant anoxia might depress the vitality of the secretory cells. All these factors might be expected to reduce the flow of saliva, and experiments carried out at Harvard by Gregersen and Bullock show that in anhydræmia the flow is in fact reduced. On the other hand, Kirk, writing in a journal not received in this country, has been unable to correlate the plasma chloride concentration or the degree of dehydration with the condition of the tongue; he believes that dryness of the tongue is better correlated with the degree of intestinal motor paralysis. Let us examine this question, remembering that examinations of the plasma are of limited value in determining the state of the body fluid economy.

The plasma has been likened to a manometer on the side of a great reservoir, the reservoir being the organs and tissue spaces. The manometer tells accurately the level of fluid inside the reservoir, so long as the reservoir is standing on the level; but if it is agitated or pushed over on its side so that the manometer is on top, the manometer will be inaccurate. Similarly, in such conditions as shock and hæmorrhage the plasma contents may be greatly disturbed without much effect on the tissues. Let us first consider the experiments of Gregersen and Bullock, who demonstrated a correlation between the flow of saliva and the volume of the plasma. They were concerned with water deprivation. They found that in persons kept for two days without water, the salivary flow was reduced from an average of five cubic centimetres in five minutes to 0·7 cubic centimetre. The plasma volume fell from 3,000 cubic centimetres to 2,240 cubic centimetres, and the plasma protein level rose from 7·7% to 8·2%. The body weight fell from 88·6 kilograms to 83·0 kilograms. From these figures it may be deduced, after allowance has been made for water loss in the expired air (300 cubic centimetres per day), in the insensible perspiration (600 cubic centimetres) and in the urine (450 cubic centimetres), that the great fluid reservoir of the tissues was but little depleted during the course of the experiment—that is to say, during the production of considerable dehydration as measured by the changes in the plasma. In intestinal motor paralysis, on the other hand, a very different physiological picture is produced, in which the tissue fluids as well as the plasma are affected. Normally some seven to nine litres of fluid per day are poured into the alimentary canal by the digestive glands and an equal quantity of fluid is resorbed from it into the blood; in intestinal motor paralysis this circulation of body fluids is greatly impeded; large quantities of fluid are lost, and large quantities become stagnant in the lumen of the gut. It is said that signs of dehydration are not conspicuous till the amount of fluid lost is about four litres—that is to say, more than the total volume of the plasma. It is clear, then, that when the body is deprived of water the plasma is depleted before the tissue fluids, and it is not till the tissue fluids begin to be depleted that signs of dehydration become evident. In a state of tissue dehydration—that is to say, a state more severe than anhydræmia and including it—a more severe depression of salivation might be expected, and the observations of Kirk on intestinal motor paralysis (as they are quoted) show that it does in fact occur; the mechanism of production of dryness of the tongue is the same in *diabetes mellitus* in which the tongue is parched when dehydration has occurred through polyuria.

There is a third way in which the tongue may become dry: as everyone knows who has had a cold in the head, it may be caused through breathing through the mouth. One should not forget that dryness of the mouth does not necessarily connote suppression of the saliva. In states of intoxication it might be expected that the complex metabolic processes resulting from the activity of the cells of the salivary glands would be depressed, just as cerebral function and muscular power are impaired; in this way might be explained the common observation of "dry mouth" in the early stages of febrile illness, before there has been any abnormal fluid loss from the system.

The Coated Tongue.

The most important part of the tongue as an index of health is the epithelium over the dorsum. This is a squamous stratified epithelium, similar to that of the skin, but thinner and thrown into innumerable papillæ. There is no need here to describe the epithelium of the tongue in detail, but of these papillæ the filiform papillæ, which are far and away the most numerous and distributed thickly all over the dorsum, are thin, barb-like projections and are relatively opaque, in a similar fashion—but in a less degree—to those of the corium, which make the skin appear white; they give the tongue its translucent pink colour, in contrast to the redness of the lips and buccal mucosa. A continual proliferation and shedding of the squamous epithelium of the tongue and its papillæ are taking place, just as in the squamous epithelium of the skin. The epithelium is responsive to many metabolic changes; when it proliferates rapidly each filiform papilla appears whiter and thicker, or when it is less readily shed and the filiform papillæ are longer than normal, the tongue takes on a coated appearance.

The older writers used to classify the coated tongue into several degrees of coatedness. The tongue was described as a "stippled tongue" when it was sprinkled with white specks, practically confluent, due to a trifling excess of epithelium on the papillæ either from overgrowth or from insufficient removal. The tongue was described as a "coated tongue" when the intervals between the papillæ were filled up and a continuous coat was presented; two

varieties of the coated tongue were the "strawberry tongue", in which injection underlies the coating and the red fungiform papillæ project, and the "plastered tongue", which looks as if white lead or mortar had been carefully spread upon it, abruptly ceasing at the edges. The minute anatomy of the plastered tongue is that of the ordinary coated tongue, only more so—the elongation of the papillæ is greater, the intervals are more filled up and more miscellaneous material has accumulated on the surface. The tongue was described as a "furred tongue" when the papillæ could be seen by the naked eye to be greatly elongated, perhaps up to one-eighth of an inch, so that the surface looked as if covered with coarse, matted fur. The elongated papillæ are often tipped with brown and more or less obscured by incrustation. The tongue was described as an "incrusted tongue" when the elongated papillæ were separated and covered over by a brittle crust; this largely consists of microbes together with detached epithelium and extraneous and miscellaneous material. This crust may even break off and look like a cinder.

Now what is the cause of the abnormal proliferation of the papillary epithelium or its retention which produces the coating on the tongue? W. H. Dickinson, to whose writings on the tongue in 1888 and 1903 many subsequent writers have been indebted, attributed it to pyrexia. In his own words: "Cell growth increases with the temperature, physiologists tell us, up to about 104° F. and diminishes as it passes this point. The overgrowth of epithelium occasioned by pyrexia is the chief though not the only agent in coating the tongue in recent and acute disease. . . . We may regard the mouth as a hot-house for the cultivation of epithelium on the tongue and the tongue as a thermometer. . . . Want of friction, scour and wash occasioned by deficiency of saliva is a secondary factor in producing the coated tongue."

This was written in 1903 and was orthodox doctrine at that time. We must remember that forty years ago the regular measurement of the body temperature of sick people with a clinical thermometer was by no means a generally practised routine. Today the "hot-house" theory could never be put forward; everybody knows that the tongue is frequently heavily coated when there has been no pyrexia and no disuse, especially in alcoholic intoxication, in cirrhosis and neoplasia of the liver, in acute psychosis and in acute appendicitis, in uræmia and in intestinal obstruction.

Before considering what alternative explanations may be offered for the formation of coat on the tongue, let us examine some quite recent literature on the subject. Jeghers states that "the coated tongue has probably occasioned more concern in the layman's mind than in medical circles: a slight coat on the tongue is a common normal finding, especially on the less mobile posterior portion". Cabot and Adams, in their text-book of physical diagnosis, make the following statement:

> Coated tongue (due mostly to lack of saliva) is rarely of value in diagnosis and there is no need to distinguish the varieties and colour of coats; but a few suggestions may be obtained from it. Many persons who seem otherwise perfectly healthy have coated tongue in the early morning. This is especially true of mouth breathers, smokers and those who keep late hours. In those whose tongues are usually clean, the appearance of a coat may be associated with digestive disturbance, constipation or fever. In a patient with digestive disturbance a clean tongue often means peptic ulcer.

Comroe states that "many persons have a coated tongue in health, especially smokers". Prinz and Greenbaum make the surprising statement that various types of coated tongues may be seen in more than 50% of normal people in middle life; with advancing years coated tongue is less frequently observed.

To summarize these authors' statements, a coated tongue is frequently present in healthy, normal persons, especially in smokers, especially in the mornings, and especially after late nights; it may also be associated with constipation.

It is not true that constipation causes coated tongue; it is important to dispel this fallacy, for the proprietors of some aperient medicines by extensive and costly advertising seek for their own profit to make the public and medical profession believe that it does so. In healthy persons whose bowels have been confined for periods of two, three and four days—for instance, in women who have sustained laceration of the perineum in childbirth—I have many times observed a perfectly clean, moist tongue. It often happens that constipation is a symptom of a febrile illness or other toxic disease; in such a case a coated tongue might be expected.

I believe that the one common factor of all the conditions in which there is overgrowth of the filiform papillæ is a state of intoxication, or in medical parlance "toxæmia"; in fact, I think it highly probable that the so-called overgrowth of the filiform papillæ may be analogous to cloudy swelling. Intoxication may be either exogenous or endogenous, either organic or inorganic, either bacterial or chemical.

Alcohol is an example of an exogenous chemical toxin. It does not produce intoxication unless it is taken into the body in a dosage in excess of the amount which can be at once metabolized, which varies in different persons.

> A patient of mine, a lady of about fifty years, drinks alcoholic liquor in large quantities in order to gain relief from distressing emotions from which she cannot otherwise escape. Usually she is outwardly well even when she has taken a lot and her tongue is clean and moist. One day she drank much more than usual and fell into a stupor; I saw her in this state and observed that her tongue was coated.

Examples of bacterial intoxication are frequently encountered. This form of intoxication is to be regarded as exogenous. It may be either local in its effects or general. When a bacterial toxin has a selective action upon a particular organ of the body, as in diphtheritic myocarditis or peripheral neuritis, the tongue is not usually coated; in sapræmia, on the other hand, when the intoxication is general, the tongue is usually coated.

An example of endogenous intoxication occurs in uræmia, which nearly always produces a coated tongue. In acute psychosis also a state of intoxication is common; it is recognized by many visceral symptoms resembling those of bacterial sapræmia, and among them a coated tongue is conspicuous. In neurosis, "staleness" and chronic fatigue there are often many symptoms of visceral disorder, including symptoms closely resembling those of intoxication, such as langour, headache, unpleasant taste in the mouth, distaste for breakfast. A coat at the back of the tongue is frequently seen in persons suffering from neurosis. Disordered or disorderly mental activity appears to produce (or to be frequently associated with, or perhaps even to be due to) a toxic disorder of metabolism, of which one symptom is the coated tongue.

If overgrowth of the filiform papillæ associated with intoxication is a general cause of coated tongue, an impediment to the normal shedding of the epithelium of the papillæ is a contributory factor. Mackenzie, in his book on "Symptoms and their Interpretation", states that "the principal cause of a furred tongue is absence of friction, usually due to deficiency of saliva and insufficient mastication. A person who has no appetite has a furred tongue because he does not masticate. A person who bolts his food or lives on slops has a furred tongue from the same cause". I believe that Mackenzie overlooked the possibility of a toxic factor in the examples which he quotes; a deficiency of saliva, lack of appetite and a diet of slops all suggest the possible presence of toxic disease as well as lack of mastication. The occasional occurrence of a unilateral coat on the tongue—it is said to occur in trigeminal neuralgia and hemiplegia—supports the view that want of friction may so far prevent the normal shedding of epithelium as to lead to coated tongue. I have never seen a unilaterally coated tongue.

The Denuded Tongue.

Raw Tongue.

A process corollary to overgrowth of the filiform papillæ is excessive desquamation of the lingual epithelium. This may occur in patches when the tongue has been heavily coated, leaving the granular layer exposed so that a raw

red area is present, which is sore. The raw area soon becomes covered with a very thin epithelium, almost like that which spreads over the granulation tissue from the edges of a wound, and the soreness abates; but the delicate new surface is supersensitive and also easily damaged, so that the soreness easily returns. A somewhat similar condition may affect the whole surface of the tongue at once without its having been previously coated, so that the tongue may resemble raw beef and be very sore. This condition of the tongue is sometimes found in untreated pernicious anæmia (Hunter's glossitis). The administration of liver extract causes a rapid return of the epithelium to its pristine state, papillæ and all. The condition is clearly the result of deficiency from the body economy of some factor contained in liver extract.

Marginal exfoliative glossitis (Möller's glossitis) is a denudation of the tongue—at the margin as its name implies—down to the granular layer, so that the affected part is red and sensitive and sore. The condition is confused by many writers with Hunter's glossitis, but it looks quite different; the area is not so smooth and the epithelium covering it is very delicate. The condition does not respond to injections of liver extract or to treatment with aneurin or riboflavin or nicotinic acid. In my experience it occurs nearly always in women of middle age who have been subject to unusual nervous tension. I regard it as analogous to dermatosis. I have treated it successfully with small doses of stilbœstrol and measures directed to the relief of nervous tension; but I do not suggest that such treatment is specific.

Geographical Tongue.

Geographical tongue, also known as wandering rash of the tongue, starts as a small, whitish, rounded, somewhat raised area on the dorsum of the tongue near the front, in which the epithelium is thickened. It soon breaks down in the centre, the thickened most superficial layer of the epithelium being shed. It is now bounded by a white, irregularly circular, linear margin formed by the thickened epithelium at the periphery. This is called a geographical patch from its map-like contour. It is not sore. It may remain like this unchanged for many weeks, the epithelium in the centre growing up normally, or it may move or migrate rapidly. Other similar spots may appear and coalesce with it. A new spot may start within the circumference of an old geographical patch. The causes of the condition are unknown. It occurs at any age. Most writers dismiss it as a harmless abnormality for which no treatment is required. I believe it is usually a sign of impaired health. I have never seen it in anyone who was robust and very well, but it is not uncommon in persons suffering from rheumatism, neurosis, nephritis, tuberculosis and chronic catarrhs.

Mucous Patches of the Tongue.

Mucous patches of the tongue are not a bit like geographical patches. They are at first round, slightly raised red spots from the size of a large pin's head to the area of a threepence. They are irregular in outline. Soon the epithelium over them becomes macerated and whitened and loosened, so that the centre is occupied by a whitish follicle, and around this is a brick red, slightly raised, velvety border, which is sharply defined.

The Smooth Tongue.

The smooth tongue is not the same as the denuded tongue; its smoothness is due to atrophy and not to shedding of the papillated epithelium. It stands to reason that a smooth tongue cannot be coated. I think that some confusion has arisen in writings on the smooth tongue from the fact that atrophy and denudation of the epithelium often coexist—a state of affairs to which the term atrophic glossitis has been applied. I think denudation is a term preferable to glossitis; although a superficial inflammation is usually present, it is secondary. Atrophy and denudation, though they may coexist, appear to arise from different causes.

It has been shown by Oatway and Middleton that smooth tongue seldom accompanies normal or excessive gastric acidity, but that it commonly accompanies achlorhydria. Still, no rules can be laid down relating the smoothness of the tongue to the gastric acidity. One patient of mine, whose gastric acidity was excessive to a quite unusual degree, had a smooth tongue, and one not infrequently sees persons with achlorhydria whose lingual papillæ are normal.

There is no evidence that a smooth tongue results from nutritional deficiency, nor any evidence that a smooth tongue can be made to grow normal papillæ by the administration of any particular foodstuff or food factor. The administration of hydrochloric acid is certainly ineffective.

On the other hand, in many of the diseases in which both atrophy and denudation of the tongue occur, such as pernicious anæmia, sprue, hypochromic anæmia, pellagra, ulcerative colitis, chronic pancreatitis and steatorrhœa, gastric subacidity is common; but there is also a state of deficiency, due to internal or external causes, which may be remedied by the administration of the food factor or factors that are lacking. Deficiency of the vitamin B_2 complex appears to be common to all these diseases and supply of it is said to remedy the sore tongue very quickly. Whether these substances will cause restoration of atrophic lingual papillæ to their pristine normal state, as well as restoring the denuded lingual surface to its papillated state, is not related by writers on the subject, and I doubt whether they will do so.

Other Abnormalities of the Epithelium.

Fissured Tongue.

Fissured tongue (or scrotal tongue) is a congenital anomaly found in about one out of every 200 persons and present in minor degree in many more. It also occurs, along with enlargement of the tongue, in mongolism. In most persons with fissured tongue there is excessive gastric acidity. Fresh fissures may be observed to form on the tongue during adult life, from breaches in the epithelium which subsequently heal.

Small Ulcers of the Tongue.

Small ulcers of the tongue may result from contact with rough teeth or dentures, or from herpes. Sometimes in persons with excessive gastric acidity minute shallow ulcers are seen on the dorsum of the tongue near the margin and are called dyspeptic ulcers. Their presence in my experience usually coincides with the occurrence of relapse of symptoms, and indicates that medical treatment is either required or is not satisfactory.

Scarred Tongue.

Scarred tongue may indicate epilepsy, the scars having been caused by the teeth during a fit.

Other Abnormalities of the Epithelium.

Other abnormalities of the epithelium are seen in leucoplakia of the tongue, in neoplasia, in chancre, in gummatosis, in *lichen planus*, in median rhomboid glossitis and other disorders, which are mentioned in books on the tongue and on oral pathology, and which I do not propose to include in this short paper.

The Unsteady Tongue.

Some persons are unable to protrude their tongue steadily. In some nervous diseases there are peculiar kinds of unsteadiness of the tongue, as in chorea and Parkinsonism. In general paralysis of the insane and in the prodrome of *delirium tremens* the coarse tremor of the tongue and lips is characteristic. The fibrillary tremors of the tongue in nuclear or infranuclear hypoglossal paralysis are also characteristic. Apart from sufferers from these nervous diseases, there are many persons whose tongues exhibit a marginal tremor when protruded; sometimes these persons also exhibit a flutter of the lightly closed eyelids. What is the interpretation of this? Is it a morbid sign or is it compatible with health? There is no doubt that robust young people in good health may exhibit tremor of the tongue at times of emotional stress,

and a visit to the doctor may be a sufficient stress to produce lingual tremor. Nevertheless, there are many whose tongue is continually tremulous, even when no signs of nervous stress are in evidence, such as rapid pulse rate and a strained manner. Neurosis is generally the cause of this lingual tremor, and the physician should always bear it in mind; it may explain many visceral symptoms. The mechanism of the production of fine tremor is unknown.

Conclusion.

One could not sum up the things that matter most about the tongue in medical diagnosis more succinctly than in the words of James Mackenzie in his book on "Symptoms and their Interpretation": "In health the tongue should be evenly and steadily protruded, moist and of a slightly translucent pale red colour." I have endeavoured to show how and in what circumstances it may depart from this healthy state.

A PLEA FOR THE STANDARDIZATION OF THE LEPROMIN TEST.

By JOHN W. FIELDING,
School of Public Health and Tropical Medicine, University of Sydney,

AND

ROBERT G. COCHRANE,
Lady Willingdon Leprosy Sanatorium, Chingleput, Madras, India.

LEPROSY and the lepromin test need much investigation before a state of clarity is reached. H.W.W. (1941) states that: "Just as the disease itself presents problems that are unique, so is this reaction unique among immunological skin tests." Muir (1934) initiated the standardization of the test itself, while Wade (1939) attacked the problem of designation, which had been in a state of confusion. He concluded that the most appropriate title was that given in the title to this paper. Wade (1941) apparently recanted, for he made the following observation: "It therefore seems useful and appropriate to apply to it the name of the originator of the test in leprosy, as the 'Mitsuda-type reaction' or the 'Mitsuda phenomenon'." Apart from these suggestions, the test has been variously named—Mitsuda skin reaction vaccine test for leprosy, leprin test, leprolin test. Whatever is the final outcome of the controversy on the designation, there can be little doubt that permanency of procedure in preparation and uniformity of the strength of bacillary content are of the utmost importance.

CELLULAR-BACILLARY OR BACILLARY ANTIGENIC ACTIVITY.

With regard to the question of the reactive element, there are two schools of thought. The first, which includes Stein and Steperin (1934), Fernandez (1939) and Dharmendra (1942), considers that the antigenic activity is a property contained in the bacilli. The second school is of the opinion that the responsible agent for the reaction is contained in both the cellular and the bacillary proteins. Rodriquez (1938) is most emphatic on this point. In the preparation of lepromin, deposition of the ground-up tissue is of short duration (ten minutes); consequently there is a high concentration of suspended tissue element. We suggest that this, rather than assisting the reaction, has a retarding influence; such an influence may, however, be minimized somewhat by prolonged tissue grinding. This frees the organisms and thereby leaves them ready for more instant action. It would appear that in a given quantity, the actual tissue element is reduced as the bacillary content is increased. In Table I it will be noted that 0·4 gramme of human tissue (number 1) when made up with 100 cubic centimetres of saline solution contained 1,800,000 organisms per cubic centimetre, while 0·4 gramme of rat tissue in the same amount of saline solution (number 13) and under the same conditions of grinding contained some 312,000,000 organisms per cubic centimetre. We endeavoured to produce a tissue-free lepromin to solve the question; however, Dharmendra's (1942) simplified bacillary lepromin ended our quest. His results, when taken in conjunction with animal experiments of the senior author of the present paper, appear to prove that the reaction is one of bacillary protein alone.

Antigenic Activity and Acid-Fastness.

Agreement on the question of antigenic activity and acid-fastness appears to be lacking. Hayashi (1933) somewhat vaguely holds that loss of acid-fastness of the organisms in

TABLE I.

Lepromin Number.	Tissue.	Lepromin Technique.	Amount of Tissue.	Carbol-Saline Solution. (Cubic Centimetres.)	Count Technique.	Microscopic Factor.	Number of Fields Counted.	Amount of Field.	Number of Organisms per Field. (Average.)	Number of Organisms per Cubic Centimetre.	Remarks.
1	Human.	Muir.	0·4 gramme dry.	100	Breed.	300,000	18	Whole.	5·74	1,800,000	Grinding extra.
2	Human.	Muir.	0·2 gramme dry.	100	Breed.	300,000	9	Whole.	49·3	15,000,000	Grinding slight.
3	Human.	Muir.	0·4 gramme dry.	50	Breed-Owen.	400,000	3	2 squares wide.	300	120,000,000	Grinding fair.
4	Human.	Muir.	0·2 gramme dry.	200	Breed.	300,000	12	Whole.	23·4	7,200,000	Grinding slight.
5	Rat.	Mitsuda.	0·5 gramme wet.	100	Breed-Owen.	400,000	4	2 squares wide.	1,010	404,000,000	Grinding fair.
6	Rat.	Muir.	0·1 gramme dry.	100	Breed.	300,000	3	Whole.	77	23,100,000	Grinding extra.
7	Rat.	Digestion.	Portion of deposit.	100	Breed.	300,000	Series.	Whole.	70	21,000,000	Alkaline pancreatin.
8	Rat.	Muir.	0·2 gramme	150	Breed-Owen.	400,000	Series.	2 squares wide.	750	300,000,000	Grinding extra.
9	Human.	Dharmendra.	0·5 gramme wet.	75	Breed.	300,000	Series.	Whole.	20	6,000,000	Grinding light.
10	Rat.	Dharmendra.	0·3 gramme wet.	75	Breed.	300,000	Series.	Whole.	50	15,000,000	Grinding light.
11	Rat.	Ulcer washing.	1·0 cubic centimetre.	50	Breed.	300,000	6	Whole.	9	2,700,000	
12	Rat.	Muir.	0·2 gramme dry.	100	Breed-Owen.	400,000	Series.	2 squares wide.	400	160,000,000	Grinding light.
13	Rat.	Muir.	0·4 gramme dry.	100	Breed-Owen.	400,000	Series.	2 squares wide.	780	312,000,000	Grinding light.
14	Rat.	Muir.	0·8 gramme dry.	100	Breed-Owen.	400,000	Series.	2 squares wide.	1,180	472,000,000	Grinding light.
15	Rat.	Muir.	0·2 gramme dry.	75	Breed-Owen.	400,000	Series.	2 squares wide.	750	300,000,000	Well ground and settling for 24 hours before being counted.
16	Rat.	Mitsuda.	0·5 gramme wet.	50	Breed-Owen.	400,000	Series.	2 squares wide.	2,250	900,000,000	

no way influences antigenic activity. Nagai (1938) makes a similar claim after the partial destruction of acid-fastness by means of lecithin. Kitano and Inoué (1941), on the other hand, find that destruction of acid-fastness with an ultra-supersonic wave also weakens antigenic activity of both human and rat lepromin.

That acid-fastness of a mycobacterium is destroyed by treatment with hot acid, has been shown by Hayashi and also by Fielding (1934); the latter holds that primary alkaline treatment brings about a stabilization and binding of this property. Later, the senior author of the present paper noted that loss of acid-fastness might be brought about by prolonged boiling in distilled water. Consequently it is considered that, since the nodular material was prepared by boiling as in Mitsuda's technique, the emulsions used by Hayashi were in a similar condition as regards acid-fastness, and that therefore no actual difference in antigenic properties of the two could be expected. Since Kitano and Inoué held that destruction of acid-fastness and antigenic activity ran parallel, it was thought that lepromin made from tissues fixed in an alkaline medium would have enhanced reactive properties. To test this, a comparison was made of two emulsions made by Muir's technique, one from a fresh human nodule and the other from a human nodule fixed in an alkaline medium. The former gave negative results when applied to two men, and the latter gave Mitsuda-type reactions measuring 6·0 and 5·0 millimetres in the same cases. Animal experiments carried out with Stephansky emulsions treated in a similar manner gave six positive results with fresh material, while the alkaline material produced nine positive results out of 23 simultaneous injections; the size of the reactions in these cases was similar.

These results, viewed from the standpoint of uncontrolled bacillary content, appear advantageous to the alkalinized lepromin. It should be noted that the fresh human material was only one-tenth of the bacillary strength of alkalinized material. To a lesser degree the fresh rat lepromin was at a similar disadvantage. This, of course, could have been remedied, had actual counts of the number of bacilli been made at the time of the tests.

Preparation of Lepromin.

The emulsions for the lepromin test may be made in three ways. Originally it was the practice to grind the fresh nodular material immediately after boiling it in water or saline solution. The followers of this practice include Mitsuda (1923), Hayashi (1933), Stein and Steperin (1934), Rodriquez (1938), and Fernandez (1939). The second method was introduced by de Langen (1929), and was apparently put on a more permanent basis by Muir (1934). They preferred to boil the nodules and cut them into small pieces, then dehydrate and powder the tissue before making the emulsions. However, no serious attempt to compare the two methods appears to have been made by simultaneous inoculations. Our attempts to produce a tissue-free lepromin by digestion were not so successful as were those of Dharmendra. He found that grinding of the boiled nodular material with chloroform, treatment with ether, centrifugation and emulsification with carbol-saline solution produced a pure bacillary emulsion. Also he found that with such an emulsion the reactions in man could be read in a matter of days instead of weeks. According to experiments by one of us (J.W.F.), this claim appears to be justified. Hansen lepromin and Stefansky lepromin have been tested simultaneously in animals; such tests show that the readings must be made inside of two or three days to obtain maximum results. Dharmendra has drawn attention to the necessity for extensive grinding of the tissue; to this suggestion we subscribe. Attention is also directed to the fact that the grinding of Muir's dry powder with chloroform is not so satisfactory as the grinding of wet tissue.

Standardization of Lepromin.

Comparatively little attention appears to have been given to the actual bacillary content of the lepromin, the strength being gauged by the amount of tissue (wet or dry) and saline solution. Standardization of the bacillary content of lepromin appears to have been first mooted by Muir, although Kingsbury (1931) appears to have counted the number of organisms in his emulsions used for experimental inoculations. Muir introduced a platinum loop of standard size for transferring the emulsion to a slide and thereafter spreading it over a specified area, staining and roughly estimating the number of organisms in a number of fields. Rodriquez stresses the importance of retests on doubtful or weakly positive reactors. He states that, in spite of precautions, standardization by counting the bacteria of lepromin made from various sites of the same patient cannot guarantee equality of activity. Rotberg (1939) is equally critical of these inequalities. However, these ideas are purely theoretical, since neither author appears to have made any comparisons.

Dharmendra's new scheme will undoubtedly obviate these objections. It is considered that even in this test a system of actual control of the number of bacteria will have a beneficial influence and make for more uniform results throughout. Since Dharmendra's emulsions contain bacilli that are entirely free, spreading of the films will be more uniform, with incidental ease in counting the organisms. After testing various counting schemes on many emulsions, we came to the conclusion that for counts of a low bacterial standard the Breed (1911) milk count could be adapted; it gave a fair approximation of the number of organisms present in well-emulsified lepromin. It should be appreciated, however, that pipettes standardized for milk are not necessarily accurate for emulsions of the character under consideration. Nevertheless, comparisons of milk and lepromin emulsions having been made, it is considered that the measurements are accurate enough to justify their use in the lepromin test.

When the suspensions are of a high bacillary content, we suggest a method combining the Breed count with the Owen (1923) dust count. This simply means the use of the Breed apparatus together with the special squared ocular micrometer. The organisms are thus confined to a specified area in small spaces which make for easy counting of a portion of a number of fields. The combination may be adapted for all counts, whether the bacillary content is low or high.

Breed-Owen Count Requirements and Procedure.

The usual requirements for the Breed bacteria count as used for milk are as follows: a microscope equipped with one-twelfth inch oil immersion lens and oculars, a stage micrometer, a pipette of 0·01 cubic centimetre capacity with thick wall and a teat, a guide card with a square centimetre hole, a bakelite square and a platinum wire or a needle for spreading the emulsion. For the Breed-Owen combination the extra equipment required is the squared ocular micrometer introduced by Owen. The procedure and examination in both cases are simple. These may be carried out in a few minutes, and within limits give reasonably accurate results. If a series of emulsions are to be counted, the slides should be numbered with a glass marker and placed over the guide card. The emulsion should be thoroughly agitated so that the organisms will be well dispersed. The solution is then drawn into the Breed pipette past the 0·01 cubic centimetre graduation mark, the exterior is wiped with a clean cloth and excess fluid to the mark is absorbed; the fluid is deposited on the slide and spread carefully and evenly with a needle over the area of the hole in the guide card, and the bakelite and slide being kept flat, transferred to the incubator (37° C.) for drying. Fixing and staining by Ziehl-Neelsen stain are carried out in the usual manner.

Method for Breed and Breed-Owen Calculations.

The diameter of the field for the Breed count or the side of the micrometer square for the Breed-Owen count is determined by the use of the stage micrometer. The area of the whole or squared fields determines the value of the microscopic factor (*M.F.*) or multiplier of the average number of organisms per field.

Breed Count.—In the Breed count the actual number of organisms per cubic centimetre may be calculated by the formula $M.F. = \frac{xy}{\pi r^2}$, where $x = 100$ square millimetres (the area covered by 0·01 cubic centimetre of suspension) and $y = 100$ (the number of 0·01 cubic centimetre portions in one cubic centimetre). With a 6·4 diameter ocular, a diameter of 0·206 millimetre is obtained by adjusting the tube length; the radius is thus 0·103 millimetres and the microscopic factor is found to be 300,000. The area of the field may be varied by the use of other oculars or of an ocular disk with an inscribed circle; a 10 diameter ocular will give a diameter of 0·160 millimetre with a microscopic factor of 500,000. A diameter of 0·146 millimetre equals *M.F.* 600,000 and 0·178 millimetre equals *M.F.* 400,000 and so on. When an average of the number of organisms per field has been obtained from a count of a series of fields, that number multiplied by the microscopic factor for the particular magnification gives the number of organisms in each cubic centimetre of the emulsion. Since the objective is uniformity of results, and since it is thought that this may be attained by a more uniform concentration of the organisms injected, control may be effected by concentration of emulsions of low bacillary value and by dilution of those of high value. From the figures obtained in these counts, calculations will indicate the amount of diluent to be added or discarded to bring the emulsion to a uniform standard.

Breed-Owen Count.—In the Breed-Owen count the first necessity is to arrive at a microscopic factor; since the Breed pipette delivers 0·01 cubic centimetre of the emulsion, which is spread over an area of one square centimetre, a film of 0·01 centimetre is provided. By means of a stage micrometer the microscopic field is adjusted to measure 0·016 centimetre of one side of the squared ocular micrometer; thus the area is $0{\cdot}016 \times 0{\cdot}016$ square centimetre, and the volume is $0{\cdot}016 \times 0{\cdot}016 \times 0{\cdot}01$ cubic centimetre, or 256×10^{-6}. In one cubic centimetre there are therefore $\frac{1}{256 \times 10^{-6}}$ of these, or 4×10^4; so the microscopic factor is 400,000. This figure multiplied by the average number of organisms per field gives the number contained in one cubic centimetre of fluid. When a large number of organisms are present in solution, two rows of squares are counted in various parts of the film, and the results are averaged; since there are ten rows of squares, the result is multiplied by five and the necessary calculations are made in accordance with the scheme shown above.

Lepromin Bacillary Counts.

Counts have been made of lepromin emulsions of both Hansen and Stefansky materials. As indicated in Table I, they have been made in accordance with the directions of various authorities, and extra emulsions have been prepared by digestion or simple ulcer washings. In the case of human suspensions there appeared to be a tremendous variation in the number of organisms; but in general they were all capable of being dealt with by the Breed method. The rat material was much more uniform as regards the number of organisms. These emulsions were so heavily charged with organisms that it was impossible to deal with them, even when the amount of tissue was reduced to one-quarter or more of the original suggested amounts. It was because of this that the combined Breed-Owen count was introduced, and in our hands it has given reasonable satisfaction.

Results.

The series of examinations whose results are shown in Table I form only a small portion of those actually carried out by one or other of the two methods. Quite a number have been checked against one another. Checks have also been made in some cases by altering the size of the field and thus introducing a new microscopic factor for calculations. These checks have shown that the method or methods give fairly reliable results.

Summary.

From the results of this work the following conclusions have been reached.

1. The active element for the lepromin test is contained in the bacillary content of the emulsions.

2. The parallelism of antigenic activity and acid-fastness is still uncertain and must be controlled as far as organisms are concerned.

3. Effective prolonged grinding for the separation of bacilli from tissue element is all-important.

4. Standardization of the bacillary content of emulsions is important and may well be responsible for the obtaining of more uniform reactions.

5. The Breed milk count (bacteria) may be adapted for use in standardization of the lepromin when emulsions of a low bacillary content are available.

6. A combined Breed-Owen count method may be used for emulsions of low or high bacillary content.

Acknowledgements.

Our thanks are due to Professor Harvey Sutton, Director of the School of Public Health and Tropical Medicine, Sydney, for accommodation for one of us and for permission for the other to cooperate in this investigation. We are also indebted to the following for the supply of human and rat material: Dr. Muir and Dr. Ghosh, School of Tropical Medicine, Calcutta; Dr. F. G. Williams, Chief Medical Officer, Papua; Dr. F. G. Morgan, Commonwealth Serum Laboratories, Melbourne; Dr. Edgar Thomson, Royal Prince Alfred Hospital, Sydney; and Professor H. Priestley, Department of Biochemistry, University of Sydney. Finally, our thanks are due to Mr. G. Merritt for much assistance.

Bibliography.

R. S. Breed: "The Determination of the Number of Bacteria in Milk by Direct Microscopical Examination", *Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten*, Volume XXX, 1911, page 337.

Dharmendra: "The Immunological Skin Tests in Leprosy. Part I. Isolation of a Protein Antigen of Mycobacterium Lepræ", *Indian Journal of Medical Research*, Volume XXX, 1942, page 1.

J. M. M. Fernandez: "The Early Reaction Induced by Lepromin", *International Journal of Leprosy*, Volume VIII, 1940, page 1.

J. W. Fielding: "The Staining of Acid-fast Bacilli: The Effects of Fixatives and H-ion Concentration on Acid-fastness", *The Australian Journal of Experimental Biology and Medical Science*, Volume XII, 1934, page 1.

F. Hayashi: "Mitsuda Skin Reaction in Leprosy", *Leprosy Review*, Volume IV, 1933, page 159.

A. N. Kingsbury: "Annual Report of the Institute of Medical Research (Federated Malay States)", 1932, page 63.

H. Kitano and T. Inoué: "The Mitsuda Reaction by Vaccines Treated with the Ultra-Supersonic Wave", *International Journal of Leprosy*, Volume VIII, 1941, page 29.

C. D. de Langen: "Specific Skin Reactions in Case of Leprosy", *Mededeelingen van den dienst der volksgezonheid in Nederlandsch-Indië*", Volume XVIII, 1929, page 113.

K. Mitsuda: "International Leprosy Conference", 1923, page 219 (J. B. Baillière et Fil).

E. Muir: "The Leprolin Test", *Leprosy Review*, Volume V, 1934, page 83.

K. Nagai: "Effect of Storage in Lecithin on Acid-fastness and Antigenic Properties of Lepromin", *La Lepra*, Volume XXV, 1938; cited in *International Journal of Leprosy*, Volume VIII, 1940, page 132.

J. S. Owen: "Jet Dust Counting Apparatus", *Journal of Industrial Hygiene*, Volume IV, 1923, page 522.

J. N. Rodriquez: "Observations on the Leprolin (Mitsuda) Reaction", *International Journal of Leprosy*, Volume VI, 1938, page 11.

A. Rotberg: "The Reading of the Lepromin Test", *International Journal of Leprosy*, Volume VII, 1939, page 161.

A. A. Stein and M. J. Steperin: "The Specific Allergy in Lepers", *Urologic and Cutaneous Review*, Volume XXXVIII, 1934, page 860.

H. W. Wade: "Leprolin versus Lepromin", *International Journal of Leprosy*, Volume VII, 1939, page 272; "The Mitsuda Phenomenon", *ibidem*, Volume IX, 1941, page 101.

TREATMENT DURING CONVALESCENCE AFTER HEAD INJURY.

By J. ESTCOURT HUGHES, M.S. (Adelaide), F.R.A.C.S.,

Surgeon Lieutenant-Commander, Royal Australian Naval Reserve; Surgical Specialist, Royal Australian Navy.

The Problem.

How best to conduct the treatment of patients who have sustained head injuries, so that they will be able to resume their places in society at the earliest possible moment and with the fewest residual symptoms, is a matter of great concern to medical men in every branch of the profession, because all its members are involved at some time and to some extent in the care of such patients. Because so many interesting side issues develop as one considers this problem, it is necessary to define clearly the scope of this article, and for this purpose the convalescent period will be regarded as commencing with the recovery of consciousness and ending with the return of the patient to work or with his invaliding.

The rehabilitation of those who have suffered from various injuries and diseases is a favourite subject with present-day writers, and the question of rehabilitation in relation to head injuries has not been overlooked by them. Many have drawn attention to the complete lack in the past of any follow-up or attempts at the systematic rehabilitation of patients who have met with accidents involving injuries to the skull and brain. My experience has been that this lack exists, and it is a matter which calls for improvement in the future.

If this desirable improvement is to take place, the neurological, neurosurgical and psychiatric principles of the management of head-injury patients in all stages must be appreciated, and a new attitude must be adopted by those who have formerly regarded "head cases" as somewhat of a nuisance and unworthy of the same interested attention that they were happy to bestow on other surgical emergencies. As the great majority of patients who have sustained cranio-cerebral trauma are taken in the first instance to the large public hospitals, it is essential that the members of the resident medical staffs of these institutions should be made to realize their responsibilities in this respect, a task which devolves on their teachers and seniors. Most readers will remember that in their own days as house surgeons patients who had sustained head injuries, frequently drunk, dirty and non-cooperative, did not share the glamour of the patient with acute appendicitis or perforated peptic ulcer, and the passing years do not appear to have brought about a great change in this attitude.

Even if we believe that the seeds of much invalidity are sown by this lack of interest in the early stages of treatment, there can be no doubt that the seeds are brought to full flower and fruition by inadequate, and all too often uncomprehending, care in the convalescent period, although it is readily admitted that the patience of the doctor is strained to the utmost in many cases by the attitude of the patient, his relations, his friends and other interested parties. It is the purpose of this article to present current views on the care of patients during their convalescence from head injuries, and to make suggestions for improving the facilities available at present in this country.

The extent of the head injury problem may be gauged from Rowbotham's[1] statement that 80% of the 9,000 deaths which occur annually on British roads are due to head injuries, and that for each fatal case there are four or five not fatal but severe injuries which cause prolonged invalidity and sometimes lead to permanent partial or complete incapacity. In addition to these cases there is the large number of relatively minor injuries, which, however, are far from being a negligible quantity when rehabilitation has to be considered.

Munro[2] reports that in 1932 there were 112,000 cases of fracture of the skull in the United States of America, and he states that in 1938 this number was increasing.

Watson-Jones,[3] of Liverpool, in a recent characteristically sound and forceful article, makes some observations on the problems of rehabilitation, a summary of which is included here, because the general principles enunciated by him apply closely to the subject of this article. This writer states that the principle of rehabilitation was laid down 2,000 years ago by Plato, who said: "This is the greatest error in the treatment of sickness, that there are physicians for the body and physicians for the soul, and yet the two are one and indivisible." From this quotation as a text, Watson-Jones goes on to state that, although teams comprising surgeons, specialists, physical therapists *et cetera* are an essential element in rehabilitation, the medical officer in charge of a case must learn to treat the whole disorder and not only part of it, and that continuity of supervision by one man is a fundamental requirement for success. Treatment of any lesion should not end with the discharge of the patient from hospital, but with his full physical and psychological recovery. The following lines from the conclusion of this admirable article are quoted here because it is felt that every practising doctor should dwell on them and accept them as the basis of his practice in traumatic cases, and not least in cases of cranio-cerebral injury.

> We must not perpetuate the 2,000 year old error—we must not separate rehabilitation from medicine and surgery. Above all we must not train "rehabilitationists". When the surgeon himself, with the aid of every member of his team, gives the patient confidence, explains his problems, anticipates his fears, reassures his mind, and completes his treatment, then he becomes a physician of the body and the soul. He leaves the ranks of the "physicians doomed to the practice of surgery"; he joins the ranks of surgeons devoted to the practice of medicine of the soul.

The Answer.

If we accept this as a statement of our obligations in dealing with head injuries—and we cannot do otherwise—we must next turn our attention to the consideration of the basic principles on which practice is to be founded. A carefully planned scheme of convalescent care and rehabilitation can rest on only one secure foundation, and that is on an accurate prognosis, which must answer the following questions: (i) Will the patient recover? (ii) Will he be able to resume his original work? (iii) How long will recovery take? (iv) What steps must be taken to ensure recovery? The assessment of the prognosis in cases of head injury is not easy, and it is made more difficult by many factors. Of these, the lack of knowledge of the end-results of head injuries and their treatment, stated as recently as 1938 to be at an irreducible minimum, probably takes first place, but is closely followed by the related contingency that all too frequently inaccurate clinico-pathological diagnoses are made, and that treatment based on these faulty premises is directed at dealing with symptoms rather than with an underlying pathological state. Litigational factors, too, play a prominent part in confusing the issue, as does the anxiety commonly felt about head injuries, arising from the popular belief that insanity and other similar disastrous sequelæ are commonly caused by such lesions. If the patient does not initially feel this anxiety himself, in many cases he soon appears to react to the anxiety felt on his behalf by others, sometimes from irreproachable motives, but sometimes not altogether altruistically.

With these factors on the debit side, what is there on the credit side in our search for an accurate prognosis? The most important thing is to estimate the severity of the injury. It is widely accepted that the best available means of estimating the severity of an injury to the brain and the outlook as regards recovery is the determination of the period of post-traumatic amnesia. Ritchie Russell[4] advocated the use of this method as far back as 1932, and Cairns[5] accepts it as the "best yardstick at present available", and suggests that the relationship between the period of post-traumatic amnesia and the shortest time in which return to full work can be anticipated is somewhat as shown in Table I.

TABLE I.

Duration of Post-Traumatic Amnesia.	Shortest Time before Return to Full Work.
5 minutes to 1 hour	2 to 4 months
1 hour to 24 hours	4 to 6 weeks
1 day to 7 days	6 to 8 weeks
Over 7 days	4 to 8 months

Symonds and Ritchie Russell[6] stress the necessity for most carefully determining the duration of post-traumatic amnesia. They define the end-point of post-traumatic amnesia as that time at which the patient can first give a clear and consecutive account of what is happening around him. This is decided upon by careful questioning after the full recovery of consciousness and normal orientation. The definition given by these authors enables us to avoid two common errors. The first of these is to take the time of a patient's first memory of his surroundings as the end-point. This is faulty, because a further period of amnesia, often quite prolonged, may follow such an "island" of memory. Secondly, Symonds and Ritchie Russell point out that, even if at some time a patient appears to be aware of what is going on around him, this does not mean that later on he will be able to recall occurrences at this time.

A further prognostic factor of great importance is the mental constitution of the patient prior to the accident, and with this should be coupled the amount of mental incapacity and emotional disturbance caused by the accident. The first of these can be determined by the careful investigation of the patient's personal and family history, and this and the other points by psychological investigation during convalescence. In its application this factor leads us to believe that, in general, the better the brain injured, the better are the chances of recovery.

Age is important in the prognosis, in that youth is on the side of recovery, and there is a distinct falling-off in the number of good recoveries after the thirty to forty years' mark. The occurrence of post-concussional headache influences the outlook, and to this important subject further reference will be made later.

Statistically one is justified in taking a more optimistic view than has hitherto seemed possible, and Ritchie Russell,[7] after conducting a follow-up in cases of head injury in wartime England, has reached the conclusion that post-concussional symptoms are less common than was previously thought, and that the period of disablement after a head injury may be surprisingly short. He gives figures in support of his contention, and, briefly, these are that 61% of patients returned to work in two months or less, and that a further 26% returned to work in from two to six months. Many of the patients considered were service personnel, and Ritchie Russell specifically excludes compensation cases.

These, then, are the considerations which influence us in making a prognosis, and we may now turn to the principles that govern our present-day treatment of those who have been admitted to hospital as the result of a head injury.

1. Remember that the recovery of consciousness is in itself the best sign of all, and that from the moment that this happens the patient must be given an impression of cheerfulness and optimism, and by neither word nor gesture must he be made to feel that he is lucky to be alive and that he is an object of great concern. The importance of this initial psychological approach cannot be exaggerated.

2. As soon as is reasonable, communicate to the patient and his friends the prognosis in his case, taking the optimistic line, but not at the expense of being perfectly frank. If the prognosis is inaccurate, the patient will experience disappointment and possibly resentment, and his recovery will be retarded in consequence. Much patience may be required in the making of explanations, but this must not be stinted.

3. Early mental and physical activity is encouraged, in contrast to the older principle of prolonged confinement to bed in idleness.

4. Throughout his treatment the patient is to be an active participant in all measures undertaken, and he is to be made to feel that he has a share in the responsibility for his recovery.

5. Continuity of treatment by one medical officer is to be preserved as far as is practicable.

With regard to the consideration of the details of treatment, it is convenient to divide the convalescent period into two stages, as suggested by Jefferson.[8] The first of these is the period of the patient's stay in hospital, and the second is the period between his discharge from hospital and his return to work.

In the first stage, the important question arises as to how long the patient should be kept in bed. The old rule was that a patient should be kept flat in bed and inactive mentally and physically for at least two weeks and usually longer. Today, however, there is general agreement that this is unnecessary, and indeed prejudicial to a satisfactory recovery. It is suggested that the patient recovering from an injury to the head should be allowed out of bed within a few days of recovering consciousness, and that diversional measures should be commenced as soon as the patient's interest can be aroused. Even a mild or moderate degree of persistent confusion need not be regarded as a contraindication to this procedure, because it has been found that a walk and a bath often benefit patients in such a state. Cairns advocates this; but he states that "early up" will not necessarily mean "early back to work".

The diversional methods suggested include reading or being read to, the solving of cross-word or jig-saw puzzles, net-making, drawing or knitting *et cetera*. From these beginnings a patient is gradually introduced to more definitely occupational pursuits requiring more effort both mentally and physically; but the selection of the particular tasks allotted should take cognizance of a man's inclinations and aptitudes.

Amusement is also to be catered for, and in my experience it is provided freely at service hospitals, but insufficiently at civil hospitals. Patients who show a disinclination for amusement may be allowed quietness, but attempts should be made to ensure that the patient does not use such periods for unhappy introspection in solitude.

As far as possible, the life of a patient recovering from cerebral damage should be run to a time-table, so that meal periods, periods of rest and periods of occupation are wisely mixed, and so that most of the day is dedicated to some definite object. Only thus can the boredom, laziness and lack of adaptability consequent on a stay in hospital from any cause be prevented, and their prevention is of fundamental importance in head injuries. In advising this time-table principle, however, one does not mean that it should be adhered to so rigidly as to lose sight of the fact that each patient presents, and should be treated as, an individual problem. The degree to which the routine may be relaxed increases with the patient's improvement.

A good standard of hospital discipline is necessary if this schedule of treatment is to be effective, and writers from Britain are satisfied that service patients do better than civilian patients, and accept the better discipline of the former as one of the prime factors in their more rapid and complete recovery.

Physical therapy in forms which allow the patient to be the mere passive recipient of treatment has a limited scope; but graduated physical exercises, particularly those which stimulate interest and evoke a spirit of competition, fill an important place.

During the hospital stage of convalescence the psychological estimation of the patient should be carried out, and a full ophthalmic examination should be made. This latter serves two purposes: firstly, it will reveal and permit adjustment of any ocular abnormality, thus removing a cause of headache and fatigue, and, secondly, it will establish a standard, which may be of great help in the detection or evaluation of late complications.

As the first stage of convalescence draws to a close, the question will arise as to where the patient is to spend the second stage. This may be at an auxiliary hospital, at a rehabilitation centre, if such exists, or at his own home. An auxiliary hospital is not to be confused with a convalescent home, but is a place where treatment can be continued and discipline maintained. Continued discipline presents an easy enough problem for service personnel; but in the case of civilian patients we have to rely on their voluntary cooperation, and the degree to which this is given depends on the confidence won by the medical staff in the earlier stages of treatment. The patient allowed to return home will be required to attend whole time or part time at the rehabilitation centre, according to his condition; but before his discharge it is important that his social background should be investigated by the social worker attached to the hospital, and that steps should be taken to make adjustments or give assistance in this sphere, if necessary. The second stage of convalescence has been called by Jefferson "the period of hardening", by which he means that during this time a patient ambulant and relatively free from complaints, but not fit for work or duty, is "topped up" so as to be rendered fit.

The time spent in hospital will be approximately six weeks, and by the end of this time it should be possible to review conclusively the prognosis already given, if this is necessary. Treatment in the second stage is a continuation of the methods used in the earlier stage, with certain modifications. As has been indicated already, for patients from the services this stage should be spent in a service institution under discipline, where training in service pursuits is possible. Leave from these places will be less generous for some patients than for others, and treatment which curtails leave is bound to be unpopular—a fact which makes tactful handling and careful explanation necessary. This is really continuing to build on the foundation of confidence already stressed as so imperative. Gymnastics, physical training, occupational therapy and diversional pursuits are all continued, but become progressively more strenuous and more complicated, while participation in competitive organized games is encouraged. Of this increase in the complexity of the tasks set, Cairns states that the important thing is not the initial standard of a patient's performance, but the degree of improvement which follows his efforts. At all costs the man who becomes confused at a task should not be allowed to develop a feeling of inadequacy and discouragement, which can only lead to headache and irritability with retardation of his recovery. Fatigue must also be prevented. A halt should be called while the patient is still keen to do more.

Social rehabilitation also progresses in this stage, and the patient may be allowed to attend dances, concerts and moving picture shows, his reaction to which will help to gauge his progress towards full recovery. Those who are doing well will soon ask to be allowed to return to work, and this permission may be quickly granted; but the type of work permitted must be indicated clearly. Again, this is easier with regard to service people than it is with regard to civilian patients, because in the case of the former the man can be marked as being in a specific category, and this recommendation is observed. For the civilian patient the drawback is the unwillingness of employers to take men back on part-time work. This is, of course, conditioned by the compensation factor, and the employee is equally unwilling for the same reason to return to work before he is perfectly fit.

Others who progress less quickly must continue treatment and receive encouragement; but it is not good for them or for others that patients with an unfavourable outlook should be kept attending the out-patient department or rehabilitation centre after improvement ceases. It is preferable that their cases should be finalized and other arrangements made for their care.

The Post-Concussional State.

There are some complications of head injuries that are quite apparently due to organic change in the substance of the central nervous system; but two of the more troublesome sequelæ are the post-concussional state and post-traumatic neurosis. In neither of these is an organic basis discoverable, although, to quote Cairns, we must "regard the post-concussional syndrome as organic, just as syncope is; but, like syncope, it is influenced by emotional and psychoneurotic factors". It is impossible to enter into a discussion here of the post-concussional syndrome and neurosis, both major subjects; but the differential diagnosis has been considered recently by Symonds and Lewis,[9] and their article may be consulted.

The Future.

The foregoing remarks set out what is regarded, in the present state of our knowledge, as the correct treatment of those convalescing from an injury to the head. What has been recommended is the practice in some centres, and it is felt that the same service must be made widely available. It is proposed, therefore, to conclude this article by mentioning some of the methods by which we can improve the treatment of patients who have sustained head injuries.

1. Better teaching of students is a *sine qua non* of any advance. By this statement one does not wish to imply that the teaching in the past has been faulty in matters of fact, but rather that it has often lacked inspiration, because it has been given by general surgeons without special interest in, or knowledge of, head injuries. Those who will arouse the interest of students most easily will be those who are most deeply interested themselves. This is, of course, connected with the establishment of special neurosurgical clinics, a matter to which reference will now be made. Hand in hand with the improved teaching of students will go the more satisfactory attitude of resident medical officers already referred to as so desirable.

2. In the leading article in this journal of February 21, 1942,[10] the present state of neurosurgery in Australia was reviewed and the need for the establishment of special clinics and for the encouragement of neurosurgeons was expounded so clearly that it would appear unnecessary to go into the matter again here. From this leading article, however, it is painfully apparent that Australia is ill-equipped with properly fitted and staffed neurosurgical clinics. Surely it behoves the hospital authorities in all the main centres of population to provide facilities for the practice of neurosurgery and to give the encouragement necessary to those who propose to devote themselves to this exacting branch of surgery. Neurosurgery has long since won its spurs, and should no longer be relegated to the Cinderella role amongst the surgical specialties, which it has occupied in so many places owing to the preconceptions and prejudices of hospital boards and the medical staff committees which advise them. When these clinics are established, they must not be so short of beds that patients cannot be kept in them for an adequate time. The clinics must also see that a proper liaison is established between the medical officers of the clinic and the general practitioners in the area which it serves, so that these practitioners will be fully informed of the patient's condition and of the steps proposed for his treatment. Equally, when a patient who has sustained a head injury is first treated by a general practitioner, that practitioner should not hesitate to make use of the clinic for consultation and assistance. Jefferson states that, in his experience, on an average six months were allowed to elapse before patients whose condition was difficult and unsatisfactory were referred for special advice. This is far too late; it is long enough to allow a neurosis to become well established. Insurance companies, Jefferson found, are amongst the offenders who permit cases thus to drag on, and a change in their attitude is also desirable.

3. In close relation with the neurosurgical clinic will work the rehabilitation centre, which must also come into being in all medical centres worthy of the name. Furthermore, the arrangement must not be to put into water-tight compartments the neurosurgical clinic, the fracture clinic *et cetera*, and the rehabilitation centre, but to permit of the continuity of treatment which Watson-Jones so strongly

advises. Between the two clinics the necessary scientific and social welfare follow-ups can be conducted.

4. The development and advance of scientific methods are to be expected, and amongst these electro-encephalography is likely to provide data which will help to clear up some of the difficulties surrounding the assessment of head injuries and their sequelæ.

Summary.

1. The problem of the care of patients convalescent from head injuries is stated, together with some fundamental considerations regarding rehabilitation in general.

2. Prognosis is stated to be the basis on which a programme for convalescence is founded, and the factors concerned in making a prognosis are enumerated.

3. The principles and some details of convalescent treatment are considered.

4. Suggestions about the ways in which the treatment of head injuries can be improved are set out.

Acknowledgement.

The Director of Naval Medical Services, Surgeon Captain W. J. Carr, C.B.E., R.A.N., is thanked for his permission to submit this article for publication.

References.

(1) G. F. Rowbotham: "Acute Injuries of the Head", 1942.
(2) D. Munro: "Cranio-cerebral Injuries", 1938.
(3) R. Watson-Jones: "Bone and Joint Injuries", *The Lancet*, Volume I, 1943, page 172.
(4) W. R. Russell: "Cerebral Involvement in Head Injury: Study Based on Examination of 200 Cases", *Brain*, Volume LV, 1932, page 549.
(5) H. Cairns: *Proceedings of the Royal Society of Medicine*, Volume XXXV, 1942, page 299.
(6) C. P. Symonds and W. R. Russell: "Accidental Head Injuries", *The Lancet*, Volume I, 1943, page 7.
(7) W. R. Russell: "Medical Aspects of Head Injury", *British Medical Journal*, Volume II, 1942, page 521.
(8) G. Jefferson: *Proceedings of the Royal Society of Medicine*, Volume XXXV, 1942, page 295.
(9) C. P. Symonds and A. Lewis: *Proceedings of the Royal Society of Medicine*, Volume XXXV, 1942, page 601.
(10) THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1942, page 231.

A BRIEF REPORT ON THE VALUE OF THE SELECTIVE MEDIUM OF WILSON AND BLAIR FOR THE ISOLATION OF DYSENTERY BACILLI.

By T. S. GREGORY,
Captain, Australian Army Medical Corps, Australian Imperial Force.

IN a short article, Wilson and Blair[1] described a selective medium for the isolation of *Bacillus dysenteriæ* (Flexner). This contained potassium tellurite and iron alum to effect at least partial suppression of *Bacterium coli* and coliform bacilli, together with rosolic acid, to inhibit the growth of *Streptococcus fæcalis*. As described in their summary, the medium contained these reagents in a lactose-agar base. In testing this medium at the mobile laboratory to which I am attached, we failed to obtain any useful colour differentiation between colonies of dysentery bacilli and coliform bacilli, owing to alkali production by the former and acid production by the latter. The medium has therefore been slightly modified by the replacement of lactose by glucose, to encourage the growth of lactose non-fermenters as much as possible, and furthermore, the amount of tellurite added was increased by 20%. Like Wilson and Blair, we found that Flexner strains grew well in pure culture, but *Bacillus dysenteriæ* Sonne was suppressed. In addition, however, it was found that Shiga and Schmitz strains also grew well. Despite the absence of colour differentiation of colonies, the study of pure cultures and of routine plates soon enables one to detect dysentery colonies with ease. On routine plates colonies are nearly always sufficiently numerous to provide ample material for identification by direct slide agglutination tests.

Comparison of Results Obtained in the Examination of 100 Stools Containing Dysentery Bacilli.

A small series of comparative tests was made with litmus lactose bile salt agar when this mobile laboratory was located with an Australian casualty clearing station in Syria. Specimens at that time were received without delay from the dysentery ward, so that there was less chance of contamination in bed pans than is the case in a large hospital. The results of tests on twenty stools showed that the medium of Wilson and Blair had a distinct advantage in the ease of isolation of dysentery bacilli and also in the number of isolations; but the results were not tabulated.

Subsequently, an opportunity was provided for continuing the investigation when the staff of this unit was collaborating with the laboratory staff of a British general hospital in Egypt. There we were performing the routine examination of MacConkey plates sown on the previous day by the technicians of that hospital, and the hospital pathologist kindly arranged to have some specimens, chiefly those showing bacillary exudate, sown in duplicate by the same technicians on a quarter plate of Wilson and Blair medium that was provided. One hundred of the samples examined gave rise to a detectable growth of dysentery bacilli on one or other of the media used in the comparative test. There were sufficient typical colonies on some plates for testing by direct slide agglutination, whereas on others the one or two likely colonies had to be subcultured in order to provide sufficient material for further investigation. The medium which facilitated identification by direct slide agglutination should be considered to be the more selective.

The results obtained are set out in Table I.

TABLE I.
Comparison of Media used in the Examination of 100 Stools Containing Dysentery Bacilli.

Source from which Organism was Isolated.	Type of Organism.							
	Flexner.		Shiga.		Schmitz.		Sonne.	
From bacillary exudate	52		27		3		2	
From indefinite exudate	9		6		1		Nil	
Total	61		33		4		2	
Method of Detection.	Mac-Conkey Medium.	Wilson and Blair Medium.	Mac-Conkey Medium.	Wilson and Blair Medium.	Mac-Conkey Medium.	Wilson and Blair Medium.	Mac-Conkey Medium.	Wilson and Blair Medium.
Direct slide agglutination	23	48	12	30	2	1	2	Nil
Single colony selection	16	8	7	3	2	Nil	Nil	Nil
Total	39	56	19	33	4	1	2	Nil

Discussion.

It may be seen from Table I that the majority of stools tested contained bacillary exudate. The results from stools containing different infecting organisms will be discussed separately.

Flexner Organisms.—A 43% increase was obtained in the number of isolations by the use of the medium of Wilson and Blair. Moreover, with this medium more than twice the number could be easily identified by direct slide agglutination than was the case with MacConkey's medium. Of the 61 "positive" stools examined, 22 gave negative results on MacConkey's medium, but only five on that of Wilson and Blair.

Shiga Organisms.—These results are of special interest, as Wilson and Blair did not report on the value of their medium in cases of Shiga dysentery nor in experiments with pure cultures. On this medium Shiga strains were isolated from all of the 33 "positive" stools tested; this represented an increase of 74% on the number of isolations made on MacConkey's medium. All except three of the strains isolated on the Wilson and Blair medium were identified by direct slide agglutination. On MacConkey's medium we failed to isolate Shiga bacilli from 14 of the 33 "positive" stools, and in a large proportion of the successful isolations it was necessary to pick off single colonies for identification. This high degree of selectivity of the new medium is all the more interesting as our experience has always shown that far more difficulty is experienced in the isolation of Shiga strains from the stools of dysentery patients than is the case with Flexner strains. In the course of routine work we encountered specimens of bacillary exudate from which no detectable colonies were obtained on any medium, and these have not been tabulated in this report. Such results are to be expected, especially if stools sent from a distance are not properly preserved. It seems probable that, although this 74% increase has been obtained in a special small series, a 50% increase would be exceeded in a larger series.

Schmitz Organisms.—Too few Schmitz organisms were isolated to make a useful comparison. With the one specimen giving positive results on both media, sufficient growth took place on both plates to make possible identification by direct slide agglutination.

Sonne Organisms.—In only two cases in the series were Sonne organisms found; a heavy growth of the causal organism was obtained on MacConkey's medium, but none at all on the medium of Wilson and Blair.

Comment.—It may be seen, therefore, that this simply prepared medium has distinct advantages in the isolation of Flexner and Shiga dysentery bacilli from bacillary exudates, and it is reasonable to suppose that it would facilitate the isolation of these organisms from stools with indefinite exudate or in chronic cases associated with no exudate. This selective medium could be used with advantage in an epidemic of Shiga dysentery and in the investigation of special cases in which Shiga infection was suspected.

Acknowledgements.

Grateful acknowledgements are made to Dr. Dennis, of the American University, Beirut, for a supply of rosolic acid, and to Major John Dick, Royal Army Medical Corps, for his personal interest, and for facilitating the cooperation of his technical staff.

This report is published by permission of the Director-General of Medical Services, Major-General S. R. Burston.

References.

(1) W. J. Wilson and E. M. McV. Blair: "A Tellurite-Iron-Rosolic Acid Medium Selective for B. Dysenteriæ (Flexner)", *British Medical Journal*, Volume II, 1941, page 501.

(2) L. E. Rothstadt, F. Fenner and B. A. Baker: "Bacillary Dysentery Caused by Bacterium Dysenteriæ P274", THE MEDICAL JOURNAL OF AUSTRALIA, April 17, 1943, page 341.

Appendix.

During a subsequent period duplicate tests were performed on specimens of fæces submitted for routine examination, without the selection of those containing bacillary exudate, although many contained some exudate. A large number came from a small outbreak of Shiga dysentery and provided an opportunity for testing the statement made in the concluding paragraph of the foregoing report. The results exceeded expectations and are given in Table II.

TABLE II.

Medium Tested.	Numbers of Each Strain Identified.		
	Flexner.	Shiga.	Schmitz.
MacConkey—			
By direct slide agglutination ..	22	29	2
By single colony selection ..	6	15	1
Total	28	44	3
Wilson and Blair—			
By direct slide agglutination ..	41	74	2
By single colony selection ..	3	6	0
Total	44	80	2

These results show that the isolation of Flexner organisms was increased by 57% and that of Shiga organisms by 87%.

Note on the Preparation of the Medium.

The contents of a bottle filled to a 100 cubic centimetre mark with nutrient agar were melted in a boiling water bath and 0·6 gramme of glucose was added. The whole was cooled to a temperature between 50° C. and 60° C. To the bottle were then added 0·3 cubic centimetre of a 2% solution of potassium tellurite in distilled water, 1·0 cubic centimetre of a 4% solution of iron alum in distilled water and 0·5 cubic centimetre of a 1% solution of rosolic acid in absolute alcohol. Plates were then poured. The solutions used may be kept in stock at room temperature for at least two months.

Other Observations.

Dr. Phyllis Anderson, of the Department of Bacteriology, University of Sydney, informs me that *Bacterium dysenteriæ* P274, described by Rothstadt, Fenner and Baker[2] as the cause of an outbreak of dysentery in central Queensland, will not grow on the medium of Wilson and Blair. Three strains received from Major Fenner were tested, and their failure to grow confirmed Major Fenner's own observations that the medium is not suitable for the isolation of this organism.

MENINGITIS DUE TO HÆMOPHILUS INFLUENZÆ: REVIEW OF TREATMENT.

By A. G. NICHOLSON, M.B., B.S.,

Medical Superintendent, Children's Hospital, Melbourne.

MENINGITIS due to the *Hæmophilus influenzæ* or Pfeiffer's bacillus, more commonly termed influenzal meningitis, has long been recognized as one of the most serious of the diseases of infancy and childhood, but it is doubtful if the comparative frequency of the disease is so well known.

Lindsay, Rice and Selinger in the *American Journal of Pediatrics*, Volume XVII, August, 1940, state that, if tuberculous meningitis is excluded, influenzal meningitis is the most common form of meningitis in children under the age of two years. In their review of 642 cases of all types of meningitis, 100 cases or 15·6% were of the influenzal type. The following tabulation shows the distribution of their 642 cases of meningitis, the period covered being from January 1, 1924, to March 31, 1939.

Tuberculous	205 cases	(31·9%)
Meningococcal	180 cases	(28·0%)
Influenzal	100 cases	(15·6%)
Pneumococcal	73 cases	(11·4%)
Streptococcal	50 cases	(7·8%)
Staphylococcal	5 cases	(0·8%)
Due to Friedländer's bacillus ..	1 case	(0·16%)
Due to Morgan's bacillus	1 case	(0·16%)
Undetermined	27 cases	(4·2%)

Until recently, the mortality rate of this disease has been so high that a favourable prognosis has been regarded as almost impossible. A review of the cases dealt with over the past six years at the Children's Hospital, Melbourne, shows that this pessimistic outlook was not without foundation. Among the 64 cases recorded at the hospital there have been only twelve recoveries—a mortality rate of 81·3%. Table I gives the figures in more detail.

TABLE I.

Year.	Number of Cases.	Average Age of Patients. (Months.)	Recoveries.	Mortality Rate.
1938 ..	5	26	—	100·0%
1939 ..	8	14	3	62·5%
1940 ..	5	16	1	80·0%
1941 ..	14	22	—	100·0%
1942 ..	13	19	2	84·6%
1943 ..	19	14	6	68·4%

No child under the age of seven months recovered, and a study of those who did recover gives the impression that the older the child, the better the chance of survival.

Prior to 1939, the treatment had consisted of the repeated performance of lumbar puncture and drainage of the cerebro-spinal fluid. One of the recoveries in 1939, that of a female child, aged five years, responded to this method of treatment; this fact indicates that spontaneous cure is a possibility in this complaint.

In 1939 sulphonamide therapy was administered. The first patient to respond to this treatment was an infant, aged seventeen months, who received a total dose of 47 grammes of sulphapyridine, recovery being complete after an illness of fifty days' duration. The second patient was an infant, aged twenty-three months, who received a total dose of 36 grammes of sulphapyridine, recovering after forty-three days.

The relative success of sulphonamide therapy raised the hopes that the "wonder drug" would solve our difficulties in the treatment of influenzal meningitis. A glance at the figures of the following years shows that these hopes, unfortunately, were not realized. In 1942, massive doses of sulphapyridine were given, resulting in two recoveries. One female infant, aged twenty-three months, received a total dose of 193·5 grammes of the drug over a period of forty-five days, with ultimate recovery. Early in 1943 another child, aged three and a half years, was given 356 grammes of sulphapyridine over a period of three months, and the outcome was successful. Despite these heroic measures, the mortality rate still remained depressingly high.

Observations on these and other patients admitted to the hospital this year lead me to the conclusion that the sulphonamides have no specific action on the *Hæmophilus influenzæ*, but that rather they inhibit the activity of the organism, thus allowing the patient to manufacture his own antibodies, and it is this factor which finally effects a cure.

At this period my attention was directed towards an excellent article by Alexander and others in the *American Journal of Pediatrics*. These workers have investigated the disease very thoroughly, and they point out the similarity between the structure of the *Hæmophilus influenzæ* and the pneumococcus. Both these organisms possess a capsule, and the "capsular substance is the element of the organism upon which both its type specificity and its power to invade the human body depend". Typing tests by the capsular swelling technique showed the organism concerned in influenzal meningitis to be type B. Specific type antiserum was then prepared from the rabbit, and this serum, together with a sulphonamide, was used in treating patients suffering from influenzal meningitis. The results were most impressive, fifty patients being treated with a mortality rate of 26%.

Through the courtesy of Dr. F. G. Morgan, of the Commonwealth Serum Laboratories, a small quantity of specific type antiserum was made available to this hospital. Because of the small amount of serum then on hand, a certain degree of selectivity of cases was necessary, it being considered that the treatment would be of most benefit in those cases in which an early diagnosis had been made. The necessity for early diagnosis is stressed by the appearance of the thick and tenacious exudate at the base of the brain, as seen *post mortem*.

Early diagnosis is therefore essential for successful treatment, but this is a very difficult matter in influenzal meningitis, as the initial onset of the disease is ill-defined. The majority of patients present themselves with a history of drowsiness, vomiting and irritability, and in many cases there is a history also of an upper respiratory tract infection. Neck and spine stiffness is a late sign, and in babies the full or tense fontanelle is often the only positive finding leading one to a diagnosis of meningeal inflammation. The more frequent and early use of lumbar puncture is warranted in the presence of such symptoms.

Diagnosis is established by lumbar puncture and cultural examination of the cerebro-spinal fluid. A competent bacteriologist may be able to identify the organisms from a smear of the cerebro-spinal fluid, but a diagnosis is not considered proven unless a positive finding is obtained on cultural examination.

Treatment is commenced by the intravenous administration of a normal saline and glucose (5%) solution by the continuous drip method, the rate of flow being approximately two ounces per hour for the first four hours; this represents an attempt to hasten the excretion of free carbohydrate antigen in the blood. Sulphonamide is given orally during this period.

At the end of four hours, the serum is added to the reservoir of the continuous drip apparatus, and the rate is adjusted so that the serum is administered in two hours. The drip administration is then slowed down to approximately one ounce per hour. The patient's progress is checked by repeated lumbar puncture and examination of the cerebro-spinal fluid.

Up to date, only four patients have been treated by this routine method, and consequently it has not been possible to evaluate the optimum dosage of serum or sulphonamide.

The serum dosage has been calculated according to the table given by Alexander, and is as shown in Table II.

TABLE II.

Cerebro-spinal Fluid Sugar Content. (Milligrammes *per Centum*.)	Milligrammes of Antibody Nitrogen Indicated.[1]
15	100
15 to 20	75
25 to 40	50
Over 40	25

[1] One milligramme of antibody nitrogen is equivalent to one cubic centimetre of Commonwealth Serum Laboratories serum.

Although, as has been stated, the optimum dosage of sulphonamide has not yet been assessed, it appears that a much smaller dose can be given with this treatment than is advocated, for example, in meningococcal meningitis. Alexander adjusts her dosage so that 0·3 gramme per kilogram is given parenterally in the first twenty-four hours, and this is followed by 0·1 gramme per kilogram orally for seven days.

Of the four patients so treated, two recovered and two died. Of the latter, one was an infant, aged eight weeks, the total duration of whose illness was 26 hours, and death occurred twelve hours after admission to hospital. Post-mortem examination disclosed a thick green exudate covering the brain surfaces. The second patient who died was an infant, aged sixteen months, whose meningitis was complicated by gross bilateral pneumonia, and death occurred forty-eight hours after admission to hospital.

The following tables show the progressive diminution in the number of leucocytes in the cerebro-spinal fluid of the successfully treated patients.

TABLE III.

Progressive Leucocyte Counts of the Cerebro-spinal Fluid of J.S., Aged Twelve Months.

Date.	Number per Cubic Millimetre and Type of Leucocytes.	Results of Cultural Examinations.
16/6/43	1,000; 60% polymorphonuclear cells, 40% lymphocytes.	Positive.
19/6/43[1]		
21/6/43	437; 55% polymorphonuclear cells, 45% lymphocytes.	Negative.
22/6/43[1]		
24/6/43	167; 30% polymorphonuclear cells, 70% lymphocytes.	Negative.
29/6/43[1]	213; 60% polymorphonuclear cells, 40% lymphocytes.	Negative.
5/7/43	120; 60% polymorphonuclear cells, 40% lymphocytes.	Negative.
9/7/43	15 (lymphocytes).	
16/7/43	7 (lymphocytes).	

[1] Thirty cubic centimetres of specific type antiserum given.

TABLE IV.

Progressive Leucocyte Counts of the Cerebro-spinal Fluid of J.W., Aged Two Years and Two Months.

Date.	Number per Cubic Millimetre and Type of Leucocytes.	Results of Cultural Examinations.
24/9/43	1,250; 10% lymphocytes, 90% polymorphonuclear cells.	Positive.
25/9/43[1]		
27/9/43[2]	411; 96% polymorphonuclear cells, 4% lymphocytes.	Negative.
29/9/43	363; 36% polymorphonuclear cells, 64% lymphocytes.	Negative.
4/10/43	57; 38% polymorphonuclear cells, 62% lymphocytes.	Negative.
7/10/43	27; 15% polymorphonuclear cells, 12% lymphocytes.	
11/10/43	32; 2% polymorphonuclear cells, 30% lymphocytes.	Negative.

[1] Ninety cubic centimetres of specific type antiserum given.

[2] Sixty cubic centimetres of specific type antiserum given.

These tables illustrate the immediate cellular response and rapid sterilization of the cerebro-spinal fluid, and the clinical progress of the patient keeps pace with the cerebro-spinal fluid findings.

Summary.

1. A review of the treatment of meningitis due to the *Hæmophilus influenzæ* is given.

2. Spontaneous recovery can occur in this disease.

3. Sulphonamide therapy has assisted in lowering the mortality rate.

4. An outline of the treatment with specific type antiserum and a sulphonamide is given.

Conclusion.

In conclusion, whilst stress is laid on the fact that the small number of cases submitted does not permit of undue optimism, it is considered that the treatment of influenzal meningitis by specific type antiserum and a sulphonamide is an advance and holds out a brighter prognosis for the future.

Bibliography.

N. Selinger and A. Brown: "Influenzal Meningitis", *Journal of Pediatrics*, Volume XVI, Number 4, April, 1940, page 456.

J. W. Lindsay, E. C. Rice and M. A. Selinger: "The Treatment of Meningitis due to Hemophilus Influenzæ (Pfeiffer's Bacillus)", *Journal of Pediatrics*, Volume XVII, Number 2, August, 1940, page 220.

H. E. Alexander, C. Ellis and G. Leidy: "Treatment of Type-Specific Hæmophilus Influenzæ Infection in Infancy and Childhood", *Journal of Pediatrics*, Volume XX, Number 6, June, 1942, page 673.

Reviews.

UNUSUAL BONE DISEASES.

The "Medical Clinics on Bone Diseases" by Dr. I. Snapper is an unusual type of publication on an unusual collection of diseases due to endocrine disturbance or deficiency conditions.[1] The book is really a series of short monographs embracing Recklinghausen's disease, hyperparathyreoidism, parathyreoid hyperplasia secondary to other diseases, rickets (fœtal, infantile or late), osteomalacia, Paget's disease, lipoid granulomatosis (xanthomatosis), Gaucher's disease and multiple myeloma. These conditions are probably of greater interest to the specialist than to the general practitioner.

Each disease is discussed from its original discovery, and a very good historical sketch is given of the various investigations which have established its ætiology and diagnosis.

Recklinghausen's disease is considered in great detail, and the author deals with the various investigations which conclusively prove that it is due to tumour growth in the parathyreoids. Many other diseases such as osteomalacia are accompanied by hyperplasia of these glands, a purely secondary manifestation.

In parathyreoid tumours there is a gross disturbance of the calcium and phosphorus metabolism, and the author gives various tables showing the decrease in calcium and the increase of phosphorus in the serum in cases of Recklinghausen's disease. Radiographically the diagnosis between Recklinghausen's and Paget's disease is difficult, but the blood chemistry allows of definite diagnosis. X-ray and microscopic appearances are dealt with in detail. Recklinghausen's disease is cured by removal of a parathyreoid tumour, but in other allied diseases removal has no effect on the progress of the disease, for example, Paget's disease and chronic arthritis. Renal osteodystrophy and renal failure simulate Recklinghausen's disease, but the blood chemistry is entirely different. Lipoid granulomatosis (xanthomatosis) shows multiple lesions of the skeleton simulating bone cysts and giant cell tumours, but here again there are no biochemical changes in the serum. The author points out that the biopsy specimens should be taken from fresh lesions which show islands of foam cells full of cholesterol esters. Sites of predilection are the skull and pituitary fossa and are accompanied by exophthalmos, *diabetes insipidus* and infantilism. Occasionally long bones are affected, but it is not usually a final and generalized condition.

The author deals briefly with Gaucher's disease in which splenomegaly and hepatic enlargement are present. The spleen and liver show proliferation of reticulum cells and histocytes containing a lipoid known as kerasin. These cells are also present in the bone marrow. Sternal marrow puncture is recommended rather than puncture of the spleen owing to the tendency to hæmorrhage in this condition. The bone lesions are described as fairly typical, there being bottle-like swellings of the bone (especially of the lower end of the femora) with changes in bone structure and the presence of special Gaucher cells in the marrow. Multiple myeloma (first described by Bence-Jones) is characterized by abnormal fragility of bones, bone pains, Bence-Jones protein in the urine, typical rounded areas of destruction spreading from the medulla and destroying the cortex, hyperproteinæmia and myeloma cells in sternal marrow. Occasionally difficulty is found in securing a reaction to the Bence-Jones test in cases of albuminuria, but the author details the various tests for demonstrating this phenomenon.

This work should be of importance to the specialist as a book for reference in bone disease. It is beautifully illustrated by photomicrographs and skiagrams, all reproduced as photographic prints.

[1] "Medical Clinics on Bone Diseases: A Text and Atlas", by I. Snapper, M.D.; 1943. New York: Interscience Publishers, Incorporated. 11" × 8½", pp. 232, with many illustrations. Price: $10.75.

The Medical Journal of Australia

SATURDAY, APRIL 8, 1944.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE OUTBREAK OF ENTERIC FEVER AT MOORABBIN.

THE outbreak of enteric or typhoid fever which occurred last year in the Moorabbin district, Victoria, was a typical milk-borne epidemic. There was a good deal of comment in the daily newspapers and certain persons expressed anxiety about it, but the whole event was an excellent demonstration to the public of the way in which a well-informed and competent health authority can deal with such a situation. There were other lessons which an intelligent community might take to heart, and these may be gathered from a report on the incident that has been issued in the form of a brochure by the Commission of Public Health of Victoria. This report is a document of eighty pages, including an introduction and appendix; several maps are also published. That a document of such value to preventive medicine can be published at the present time when the use of paper and printing are restricted is a matter for congratulation and shows that those responsible have a commendable sense of values.

What is known as the "city" of Moorabbin is a partly rural municipality at the south-eastern corner of the Melbourne suburban area. During 1943 its population was 24,000, and 4,500 of these persons lived in the affected area. The character of the population is midway between that of an urban and that of a rural district. A peculiarity is the preponderance of young adults; this is due to the rapid expansion of the urban areas and to the entrance of young married couples from other municipalities. What is called the "explosion" of the outbreak occurred on March 16, 1943. Several cases had been notified before this. One case was notified on March 6 and another on March 14. In the former of these two cases the patient had been in hospital since February 22 and a diagnosis was made by blood culture methods on March 2. No alarm was felt until March 15, when four fresh infections were notified. Some idea of the explosiveness of the outbreak may be gathered from the fact stated by Dr. F. V. G. Scholes, Medical Superintendent of the Queen's Memorial Infectious Diseases Hospital, Fairfield, in the foreword to the report, that on March 18 no less than 69 patients had been admitted to his hospital and others to other hospitals. Here was a situation that called for prompt and energetic action. The government medical officer and the health inspector of the city of Moorabbin were both away on active service. The medical practitioner who was acting for the regular officer was a busy practitioner and clearly could not undertake the duty of investigating the outbreak, a duty which no doubt would require all his time. The acting health inspector was holding a similar position for three other municipalities. The Department of Public Health thereupon sent Dr. C. R. Merrillees to take charge of the investigation, and with him a trained nurse and a technician; the acting health inspector was released from his part-time duties with the other municipalities. Anyone who reads this report will agree that the choice of the department was wise. Dr. Scholes in the foreword states that the acumen, sagacity, energy and speed displayed by Dr. Merrillees were remarkable. At this stage it may be stated that the total number of cases reported during the outbreak was 459. Of these, 26 were not cases of enteric fever, so that the net number of cases was 433. Of the 433 patients, 388 became well, 23 died and 13 were carriers; nine were still in hospital on July 31, 1943. It is interesting to note that for the five months March 1 to July 31 the total number of cases of enteric fever occurring in the whole of the rest of Victoria was 20. Dr. Merrillees points out that at the start of his investigations it was fairly easy to eliminate factors other than milk and water. He has a section on the case against water and another on the case against milk. It was noted that the areas of the water mains seemed to coincide with the areas in which the cases occurred. Dr. Merrillees has an array of nine arguments why water was not responsible. In the opinion of Dr. Scholes one fact will convince most people—the large and small mains which serve the affected area continue through it and serve much more densely populated cities to the westward; these were not affected by the epidemic. The affected area was one of the "zones" to which milk was supplied and all the milk came from one dairy. A point of great importance was that there was in the area a milk bar which did a large trade; but this milk bar was supplied not by the dairy in question, but by an outside source. As far as could be discovered not one person served by this milk bar became affected by the disease. The dairy drew its supplies of milk from seven sources; six of these were dairy farms and one was a milk factory supplying a fifty-quart can of pasteurized milk. It had therefore to be decided whether the central dairy had a carrier on the staff or whether a carrier was to be found among the carters or on one of the six farms. Of the six farms three were classed as clean. The remaining three were suspected and one was classed as bad in every way. Dr. Merrillees points out that many factors are involved in cleanliness on farms, and the chief of these are the lack of facilities for cleanliness and the will and energy to overcome this lack. What has to be determined is the likelihood of direct or indirect contamination of the milk with fæces or urine. In the farm which was "bad in every way" the wife of the occupier, an actual milker, passed fæces from which a culture of typhoid organisms was obtained. The family concerned left the farm on March 15 and it was closed two days later. No other carrier was discovered, so that no more infected milk was produced after March 16 or 17. It was

known that all the early patients in the zone and most of those elsewhere were known to have consumed suspected milk, and no suspected milk was served in any area other than that which provided the cases.

At the outset it was stated that the Moorabbin outbreak was a typical milk-borne epidemic. It was, as Dr. Merrillees states, not a mild epidemic, but its termination, which in Dr. Scholes's opinion might have been more abrupt, was the result of careful and energetic work. In spite of its typical character the outbreak raises several questions, some of which are sufficiently important to warrant separate discussion on another occasion. No mention has been made here of sewerage installation and of the part that flies may take in the spread of enteric fever. In the present instance the mention of flies was scarcely necessary. Dr. Scholes states that if every fly in the metropolitan area of Melbourne had been concentrated at Moorabbin, their combined efforts could not have produced an epidemic such as that which occurred. The question of the prevention, discovery and treatment of typhoid carriers arises. It is a large question; though it was dealt with in 1933 in a special report by the Medical Research Council of Great Britain (see The Medical Journal of Australia, Volume I, 1933, page 715), it will be discussed again shortly in these pages. Personal cleanliness among those who work in dairies and who handle food generally, is another large subject that cannot easily be discussed. It is one thing to see that means of washing and caring for the hands are provided; it is quite another matter to secure their intelligent use. A practitioner who came to Australia some years ago from England used to tell the tale of a demonstration that was made at a model dairy before a group of medical officers of health. The buildings of the dairy were spotlessly clean, to the milking area near the cow bail no exception could be taken, machinery did most of the milking, stripping had to be done by hand. The milker was suitably clad in white, he washed his hands carefully and scrubbed them in hot water, he sat on a clean metal stool and had a sterilized pail to receive the milk, the cow's udder and the teats were carefully washed and dried; when, however, the milker spat on his hands before setting to work, he could not understand why the assembled onlookers broke into hilarious mirth. The next question that arises in connexion with this epidemic is the pasteurization of milk—this is the most important of those mentioned. The authority that first makes the public realize that it is not safe to drink raw milk will be numbered among the great benefactors of the community for all time. The last point is that the health service in regard to medical officers should be adequate. Dr. Merrillees has something to remark in this regard when he recommends that full-time medical officers should be employed in all health services. This is one of the subjects that will merit early consideration after the war has come to an end.

Current Comment.

LIVER FUNCTION TESTS.

Any work emanating from the Northwestern University Medical School and directed or sponsored by Professor A. C. Ivy is certain to win an appreciative reception throughout the medical world. Professor Ivy and his collaborator, James A. Roth, have taken up once more the discussion of liver tests and have given a very critical assessment of their value in a comparatively recent issue of the *Quarterly Bulletin* of their school.[1] These authors make a number of points very clear and the importance of these will be generally admitted. If the liver had only one function to perform, an adequate test of this activity could easily be devised; but the liver has a multiplicity of functions and no single diagnostic procedure can possibly cover the whole. Each method should therefore have its limitations defined. Again in disease there may be dissociation of function so that the liver may be working normally with respect to one function and abnormally with regard to others. Furthermore, the liver is an exceedingly adaptable and dynamic organ, varying in its activity from day to day and showing marked diversity of action between day and night. Ivy and Roth also emphasize the necessity of taking the duration of the disease into consideration. It is now generally admitted that gross obstruction to the extrahepatic bile passages always induces injury to the hepatic parenchyma, and it is also maintained that a purely hepatic jaundice affects the finer bile tubes. The regenerative processes in liver disease can appear before the general evidences of pathological conditions have abated and a small portion of regenerated liver may account for a negative result being obtained with a test such as that with galactose, despite the fact that the greater part of the organ is still in an inflammatory state. The purpose of each test should be kept clearly in mind, whether to help in the differential diagnosis of intrahepatic and extrahepatic pathological conditions, to detect subclinical liver damage, to measure the speed of recovery or to keep a watch on chronic ailments. Ivy and Roth give a critical and detailed discussion of the many recommended tests, and their conclusions are that the most useful guides in the differential diagnosis of obstructive and parenchymatous jaundice are three—urobilinogen in urine and fæces, duodeno-biliary drainage and the "intravenous galactose method". Quantitative serum bilirubin estimation is useful in detecting subclinical icterus and in studying the daily fluctuations of an established jaundice. Cholesterol estimation does not yield useful data. In jaundiced patients the "intravenous hippuric test" is an excellent indication of hepatic insufficiency provided the kidneys are normal; in conditions not associated with jaundice, what the authors name the bromsulphalein reaction gives a very sensitive clue to liver damage so long as the circulation is normal. Chronic hepatic disease can be followed by the cephalin-cholesterol flocculation test. The whole article is worthy of careful study, and minor slips which have escaped proof-reading will not detract from its value.

CONFINEMENT TO BED AFTER SURGICAL OPERATION.

In June, 1941, D. J. Leithauser and H. L. Bergo published a paper on "early rising and ambulatory activity after operation".[2] They based their remarks on 383 appendicectomies, performed in various stages of the inflammatory process. In thirteen of the cases the patients suffered from gangrenous appendicitis and were not thought suitable for early ambulatory treatment. Of the remaining 370 patients, 277 were confined to bed for only one day. Leithauser and Bergo stated that in a large percentage of cases prolonged confinement to bed after operation might be conducive to complications, and they pointed out that of the patients in their series, those who were active on the first day after operation had the most favourable convalescence. Apparently early rising after abdominal section was first practised more than forty years ago by E. Ries, who reported his results in *The Journal of the American Medical Association* in 1899. Most of his patients were out of bed on the second day.

[1] *Quarterly Bulletin of the Northwestern Medical School*, Volume XVII, 1943, Number 3, page 179.
[2] *Archives of Surgery*, Volume XLII, 1941, page 1086.

Ries was enthusiastic about the advantages of the method. D. J. Leithauser, who has written on the subject again,[1] points out that the practice advocated by Ries did not gain support until the late twenties, and then mostly in Continental clinics. As a matter of fact, Leithauser in his recent paper gives more than thirty references to the use of early rising, and they include papers from many countries, but not one from Great Britain. In his review of the literature Leithauser finds nothing but enthusiasm for the method and states that among more than 15,000 patients "subjected to early rising" only four fatal cases occurred as a result of embolism; in one of the cases there was some doubt.

In his present paper Leithauser deals with a further 464 consecutive cases in which "practically all the patients" were got out of bed early after surgical operation. Taken with his previous series, the present group, with 64 other cases not included on the first occasion, brings the grand total to more than 900 cases. Reference to some of the statistics from the second series of 464 cases will suffice to indicate Leithauser's general results. In this group there were 39 patients operated on for chronic appendicitis, 225 operated on for acute appendicitis and ten operated on for a ruptured appendix; in each of these three groups the patient was confined to bed for only one day after operation and was kept in hospital for 2·59, 2·40 and 9·40 days respectively. Twenty-five patients, submitted to cholecystectomy, were confined to bed for an average of 1·08 days and were retained in hospital for an average of 7·40 days. For 34 patients operated on for indirect inguinal hernia the figures were one day and 6·91 days. For 33 patients who had pelvic operations, including hysterectomy, the figures were one day and 6·97 days. In three instances it was necessary to readmit the patient to hospital after his discharge. One was readmitted because of abdominal pain and fever which subsided without further operation. One patient was discharged on the seventh day after operation for a ruptured appendix; he was readmitted a few days later and a cul-de-sac abscess had to be drained. The third patient was readmitted because of the development of thrombophlebitis. Only four wounds became infected, one after appendicectomy and two after operations for hernia. There were three deaths. One occurred six hours after operation as a result of a "coronary attack". The other two deaths occurred after gastrectomy, on the fifteenth and thirty-sixth day respectively. In each instance "improvement was significant" before the terminal stage. In a series of 70 herniorrhaphies there were four recurrences.

Leithauser holds that reflexes from an incision made at operation and from traumatized areas have a distinct relationship to the development of many complications. Sometimes the vital capacity is reduced by more than 60% on the first day after operation, and seven to fourteen days are usually required for the vital capacity to return to normal. Leithauser states that 50% of pulmonary complications are established in twenty-four hours and that 90% appear by the fourth day. He finds that in the presence of pulmonary complications, coughing in the standing position, if instituted during the first twenty-four hour period, will invariably expel the mucus and cause prompt disappearance of râles. Repetition of breathing exercises will maintain clear lung fields. Leithauser's explanation of the good result is that the downward pull of the abdominal contents lowers the diaphragm and that this permits air to enter the collapsed tubules beyond the plugs of mucus. Leithauser also holds that early rising has an effect on other reflex changes such as those that lead to thrombosis and inhibition of vital organs.

The first fact to be considered in any discussion on the subject of early rising after surgical operation is that in spite of the periodical discussion of the subject surgeons generally have not seen fit to adopt the practice. This alone is not sufficient to justify condemnation of the method, for the past history of surgery shows that surgeons can on occasion be of all men the most conservative, pig-headed and uncompromising opponents of new methods. In this instance the conservative attitude is based on the view that damaged tissue should be put at rest if healing of the most satisfactory kind is to be obtained. In any case it will be asked—what is the object of getting patients up out of bed and at work again in such a short period of time as that boasted by Leithauser and similar enthusiasts. If it is a question of bed shortage in hospitals there may be some justification for haste. But most doctors will agree that in these days of hurry and nervous tension the average man will quite often receive great benefit from the enforced rest and relaxation attendant on rest for two or three weeks in a hospital bed. Leithauser and those of his creed do not, we think, pay sufficient attention to the psychological aspect. Their work is valuable because it shows that early rising may be adopted in certain circumstances without damage to the patient.

[1] *Archives of Surgery*, August, 1943.

WHEAT BREAKFAST FOODS.

THOUGH certain food faddists and some public health administrators who ought to know better loudly proclaim that the inhabitants of Britain are eating a wholemeal bread, most people who have taken the trouble to examine the facts are aware that the national loaf is made from flour which represents 85% of the wheat grain. The deliberate exclusion of the bran is due to several causes, of which the most important are three in number.[1] In the first place bran acts as an irritant so that the transit of the food mass through the food canal is unduly accelerated, leading to a marked reduction in the absorption of nutriment. In the second place bran is believed to subserve human needs best in the form of milk and egg and to a lesser degree the flesh of pig and poultry. Thirdly, the bran fraction has a high content of phytic acid, and this leads, as recent researches have demonstrated with singular unanimity, to an adverse calcium balance. The various wheat breakfast foods on the market have all much in common. They are prepared by steam cooking of the whole wheat under pressure followed by baking. This double cooking completely abolishes the irritant properties of the pericarp, and this leads to a high percentage of absorption in the alimentary canal of the human consumer. In such breakfast foods all the protein, oil, carbohydrate and minerals of the wheat grain are preserved in a palatable form. Whether the phytic acid is altered by the cooking processes has not been subjected to experimental investigation. If this phytic acid remains unreduced, it will not lead to an adverse lime balance so long as milk is taken at the same time, for milk supplies lime salts in excess of what the phytic acid puts out of action. The main criticism levelled against prepared breakfast foods is that vitamins must necessarily be reduced in the double cooking. This criticism cannot be disputed, but quantitative estimation of the vitamin loss is only occasionally proffered. There has recently been published under the auspices of the Council on Foods and Nutrition of the American Medical Association a list of 34 wheat breakfast foods with their content of thiamin (vitamin B_1), riboflavin (vitamin B_2) and niacin (nicotinic acid).[2] Some of these have had their vitamins reinforced by synthetic preparations or concentrates, and it is interesting to note that in many cases the final product has a higher vitamin concentration than the parent grain; a few have been enriched with wheat germ. A survey of the figures brings home the fact that the vitamin reduction in those without reinforcement is not nearly so great as had been expected. Further research will demonstrate whether these three vitamins mentioned can be taken as representative of vitamins in general; if this is the case the widespread use of breakfast wheat cereal can evoke no misgivings, especially if milk is consumed at the same time.

[1] R. A. Bottomley: "Wheat, Flour and Bread, with Special Reference to Enriched Flour", THE MEDICAL JOURNAL OF AUSTRALIA, February 26, 1944, page 166.

[2] G. Kitzes and C. A. Elvehjem: "Vitamin Content of Prepared Cereal Foods", *The Journal of the American Medical Association*, December 4, 1943.

Abstracts from Medical Literature.

PÆDIATRICS.

Problems in Management of Rheumatic Heart Disease in Childhood.

LEO M. TARAN (*The Journal of Pediatrics*, January, 1944) discusses the problem of control and management of rheumatic infection in the childhood population. He points out that it includes all phases of childhood welfare. The medical, social welfare and educational aspects are closely interrelated, and constitute indispensable parts of a whole. The medical aspect deals with actual treatment and research. The social side of rheumatic disease deals with environmental readjustment and rehabilitation. Education of the child suffering from rheumatic disease is an integral part of the programme. Since rheumatic disease is not a notifiable disease, it lies in the main outside of the jurisdiction of hospital and health authorities. Thus, the care of these children at present is in the hands of private institutions, convalescent homes, municipal hospitals, child health agencies *et cetera*, each one of which has its own methods, unrelated in most instances to the other agencies. The total management of rheumatic fever and rheumatic heart disease thus presents a multiplicity of problems invading all aspects of human welfare. It demands an active mobilization of all possible public and private agencies concerned with the health and welfare of the childhood population. To make such mobilization effective one must find a method of coordinating all these efforts. Since such coordination does not exist in the present planning for health and welfare, a new approach is warranted. This departure from the accepted public health planning finds its origin in the fact that the disease entity under discussion does not fit into any of the traditional schemes of treatment, management and control.

Subdural Hæmatoma in Infancy.

FRANC D. INGRAHAM AND DONALD D. MATSON (*The Journal of Pediatrics*, January, 1944) present their experiences with subdural hæmatoma in infancy. They point out that the clinical picture of an adult patient complaining of increasing headache, drowsiness, blurred vision, mental confusion, occasional vomiting, or any combination of these symptoms, and giving a history of a closed head injury anywhere from a few days to several months previously, has become widely recognized as characteristic of chronic subdural hæmatoma. A voluminous literature on the general subject of this condition has resulted, and many physicians have become acquainted with this type of injury in the adult. Very little has been recorded, however, about the occurrence and management of subdural hæmatoma in infancy. It is a common condition during the first two years of life and a source of high mortality if neglected. Mental retardation is an almost uniform result of cerebral deficiency incident to hæmatoma in infants in this age group. Trauma to the head is probably always a factor in the ætiology of this lesion. The presence of systemic disease causing increased capillary permeability may materially lessen the severity of the injury necessary. The possibility of hæmatoma as a diagnosis should never be ruled out because of the absence of a history of trauma. There is no special clinical picture which is reliable in establishing the diagnosis. Generalized symptoms such as fever, vomiting, hyperirritability and failure to gain weight are frequently found alone or in addition to the more specific neurological findings of convulsions (the commonest symptom noted in the series), stupor and paralysis. Infants who show early abnormal enlargement of the head should never be abandoned as having incurable internal hydrocephalus until subdural hæmatoma has been excluded. The diagnosis can be made with certainty by bilateral puncture of the subdural space and by this method alone. A simple routine for carrying out this procedure safely is presented in detail. The rapid increase in brain volume during the first two years of life must be unrestricted to ensure the normal mental development of a child. Therefore radical craniotomy with excision or wide decompression of constricting subdural membranes is essential if cerebral deficiency is to be avoided. Infants during the first two years of life will tolerate well radical surgery for this lesion, if proper preliminary measures and supportive treatment are undertaken. The frequency with which subdural hæmatoma is found in infancy is proportional to the intensity with which it is sought. Whereas treatment is fundamentally a neurosurgical problem, suspicion of the diagnosis must rest primarily with the pædiatrician and the general practitioner.

Encephalitis Complicating Measles.

HENRY A. REISMAN AND ALEXANDER S. ROSEN (*American Journal of Diseases of Children*, December, 1943) state that during the period between January, 1936, and May, 1941, there were admitted to the Queensboro Contagious Pavilion 29 patients with encephalitis which followed acute contagious diseases of childhood. In the greatest number of these, 20, the infection followed measles. In regard to the diagnosis of this condition the authors state that the majority of patients have either a fading eruption of measles or a characteristic discoloration following the eruption. The history of measles, the sudden invasion of symptoms pointing to involvement of the central nervous system usually two to six days after the onset of the measles rash, the varied symptoms of the changes in the cerebro-spinal fluid, should point to a diagnosis of encephalitis complicating measles. In 16 of the 20 patients the spinal fluid showed an increased cell count varying between 11 and 1,260 cells. In most of the patients the fluid showed a preponderance of lymphocytes and the sugar content was either normal or increased. The protein content was normal in all except two cases, in which it was increased. The amount of chlorides in the spinal fluid was within normal limits. Two of the twenty patients died. Both of these patients were admitted to hospital in coma and with hyperpyrexia and died within twenty-four hours after admission from what appeared to be an overwhelming invasion of the nervous system. Of the 18 who survived, two were examined four years after the illness and eight one year after the attack of encephalitis. Eight of the ten patients examined were entirely normal. The other two showed definite changes in personality without mental deterioration. The incidence of sequelæ does not seem to have any relation to the severity of encephalitis. Treatment was entirely symptomatic. Sedation was used for restlessness, twitchings and convulsions. In a few cases spinal fluid was removed to relieve intracranial pressure. In the majority this was not considered necessary, the spinal tap being made chiefly as an aid to diagnosis and progress. To the patients who had more severe symptoms hypertonic medication was given, such as the intravenous administration of a hypertonic solution of dextrose. Magnesium sulphate was given intramuscularly. To those patients unable to take nourishment by mouth, liquid foods were given by gavage by means of the Levin tube. Vitamins were supplied because of the limited intake of food.

Neonatal Death.

CHARLES MCNEIL (*Edinburgh Medical Journal*, August, 1943) states that at the maternity pavilion of the Royal Infirmary, Edinburgh, there were 5,300 viable infants in the period 1939-1940. In this series there occurred 650 deaths, and of the 650 babies, 541 were examined *post mortem*. Chief causes of death were (*a*) asphyxia, (*b*) intracranial hæmorrhage and (*c*) congenital defects, for example, heart lesions, alimentary atresias, *spina bifida*, *et cetera*. It is pointed out that prematurity *per se* is not a cause of death, but is characterized by an immature condition of the body in which death is more likely to occur either at birth or in the period shortly afterwards (the neonatal period, which comprises the first four weeks of infant life). Two suggestions are made for improving the prognosis in prematurity. Firstly, to improve the general standard of maternal health so that prematurity is reduced to a minimum. Secondly, to increase the premature infant's chances of survival by improving methods of nursing and feeding. With regard to the second of these points, a plea is made by the author for the improved teaching of infant dietetics to nurses and medical students. Further, the need to impress upon them the advantages of breast-feeding and its management during the critical first weeks is emphasized.

Acquired Syphilis in Infants and Children.

GEORGE W. CRESWELL AND ELIZABETH LEECH (*American Journal of Diseases of Children*, December, 1943) report three cases of acquired syphilis in infants. They state their belief that primary lesions are often unrecognized in infants with acquired syphilis. The disease in one member of siblings should arouse a suspicion of acquired syphilis if there is no evidence of the mother having or having had syphilis. Since the risk of subsequent development of syphilis of the cardio-vascular or of the central nervous system is greater in infants with acquired syphilis than in those with congenital syphilis, emphasis must be placed on

the importance of alertness to the discovery of this type of disease. Serological tests and radiological examination should be used when symptoms are absent. All lesions should be carefully noted and followed systematically by a Wassermann test as a means of prevention of subsequent more serious difficulties. Infants in whom the disease is diagnosed early have a more favourable prognosis as to "cure". Complete studies of the families should be made in all such cases to insure prompt location of infected persons and early therapy. The number of cases of acquired syphilis in children is occasionad largely by social, moral and economic conditions in the community. Overcrowding of rooms, lack of adequate sanitation and poor economic conditions with accompanying lowered moral standards play a definite part in the spread of this type of disease. Prevention of new instances of acquired syphilis in children will result from the amelioration of these contributory factors and from the improvement of social and economic conditions in each community. Training physicians to recognize early this type of syphilis and to administer antisyphilitic therapy immediately will prevent later tragedy.

ORTHOPÆDIC SURGERY.

The Plantaris Muscle.

E. H. DASELER AND B. J. ANSON (*The Journal of Bone and Joint Surgery*, October, 1943) have made a study of the plantaris muscle by examining 750 consecutive lower extremities. They hold that the muscle is of interest from the anatomical and phylogenetic points of view, and that also it is of some surgical usefulness. The muscle and its long, slender tendon may be regarded as the vestigial remains of a primitive flexor muscle of the toes; although it was originally continuous with the plantar aponeurosis, it was later rendered discontinuous through intermediate attachment to the calcaneum. The plantaris muscle and its tendon are subject to considerable variation in both origin and insertion. In 150 lower extremities the authors encountered four types of insertion of the tendon. The plantaris muscle was absent from 50 of the 750 lower extremities examined. In one-third of the specimens from which the muscle was missing, the absence was bilateral. The plantaris tendon is particularly indicated for use as a desirable substitute for *fascia lata* in hernial repair, tendon transplants and repair of ligaments.

Trauma and Sarcoma of the Hand.

S. T. SNEDECOR (*The Journal of Bone and Joint Surgery*, October, 1943) discusses the role of trauma in the production of sarcomata of the hand. He believes that the six postulates laid down by Segond in 1907 are adequate criteria for an opinion as to whether or not trauma is the ætiological agent in any case of malignant disease. The postulates are as follows: (i) the authenticity of the trauma; (ii) sufficient importance or severity of the trauma; (iii) reasonable evidence of integrity of the part prior to the injury; (iv) correspondence of the tumour to the site of injury; (v) a date of appearance of the tumour not too remote from the time of accident to be reasonably associated with it; (vi) a diagnosis established by clinical and radiographic evidence, supported when possible by microscopic confirmation. In two cases which the author describes in detail he believes that the connexion between tumour and trauma is sufficiently clear according to these postulates; in each case a single trauma was accepted as the reasonable ætiological factor by an insurance company without recourse to legal action. The author states that tendon-sheath synovial sarcomata are rare. The diagnosis should be suspected in any soft tissue tumour of the extremities, especially if it occurs around the joints, tendons or bursæ. Usually the diagnosis is made by the pathologist after excision of the tumour; but it cannot always be made on histological grounds alone. The surgeon at the time of the excision must carefully trace the regional relationships of the tumour, so that the pathologist may have that additional information in correlating the gross tissue findings with the histological picture, and may establish the diagnosis of synovioma. The growths have been confused with several other forms of tumour. Although these sarcomata grow slowly, recur at the original site and metastasize late, their conservative treatment has given most discouraging results. Primary amputation is still the method of choice.

"Painful Feet."

R. BINGHAM (*The Journal of the American Medical Association*, January 29, 1944) states that "painful feet" developing during the period of basic training are very common amongst army recruits. In the less severe cases rest and conservative management afford relief of symptoms. In the more severe cases orthopædic treatment is necessary. These more severe lesions fall into two groups: (i) traumatic lesions of the feet, severe strains, sprains and fractures; (ii) postural foot disorders and skeletal abnormalities. At one orthopædic clinic 10% of soldiers complaining of "painful feet" were found to have Morton's syndrome (congenital insufficiency of the first metatarsal segment). The symptoms of this disorder are metatarsalgia and mid-tarsal pain caused by a congenital developmental shortening or relaxation of the first metatarsal segment of the foot. Diagnosis may be made by physical examination and verified by X-ray films. Treatment consists in the provision of an individually fitted "compensating insole", which affords a weight-bearing platform for the first metatarsal head. The author states that of 100 soldiers treated by this method, 76 have been able to continue full military duty (general service) while wearing the insoles.

Subtrochanteric Osteotomy.

J. B. KELLEY (*The Journal of Bone and Joint Surgery*, October, 1943) presents a study of 100 consecutive cases of subtrochanteric osteotomy; 59 of the patients had fused hips and 41 patients had mobile hips. The youngest patient was aged two years and the oldest thirty-two years. The author states that in cases of fused hips, the ideal position is that of 25° of flexion, neutral lateral position and neutral rotation. Patients whose hips are fused in that position have the best gait and do not complain while sitting, standing or walking. When more than 1·5 inches of shortening are present, 5° to 10° of abduction are recommended. To correct deformities at the hip joint when operation on bone is necessary, a high, curved osteotomy is indicated. Abduction, adduction, flexion and rotation can be readily corrected without the sacrifice of leg length. The position required to produce a successful result can be obtained by attention to detail. When the osteotomy has been performed, the opposite hip should be flexed, so that the lumbar portion of the spine is flattened. The extremity should be held in the desired position until the fragments are immobilized in plaster. If the surgeon is not satisfied with his clinical estimate of the position, he should make use of a gonimeter. It is difficult to immobilize the pelvis. A snugly fitting double plaster of Paris spica extending well above the nipples is necessary. The opposite hip should be in the position of abduction. When a subtrochanteric osteotomy is performed in the presence of a mobile hip, the use of the Roger Anderson pin apparatus or skeletal traction may help the surgeon to control the fragments; but the author points out that the Roger Anderson apparatus should not be used unless the surgeon is trained in the technique of its use and is aware of its dangers.

Internal Fixation of Metacarpal Fractures Exclusive of the Thumb.

E. F. BERKMAN AND G. H. MILES (*The Journal of Bone and Joint Surgery*, October, 1943) state that it is safe to treat the majority of simple metacarpal fractures, exclusive of those affecting the thumb, by the insertion of one or two Kirschner wires under local anæsthesia. The patient may be allowed to return to duty on the following day. The method has been used in 20 cases over a period of three months, without the occurrence of a case of infection and with gratifying results. The majority of fractures were simple or comminuted, involving the distal third or metacarpal neck; the remainder were oblique, involving the mid-shaft, or simple transverse, involving the base. The authors state that any of these types of fracture are ideal for treatment by the insertion of wires; single wires are used for fractures through the neck or base of the metacarpus and double wires for oblique fractures of the mid-shaft. The wire transfixes the fragments at the line of fracture. The wire is cut about a quarter of an inch from the skin margin and is sealed with collodion. A small cork is applied over the cork tip and fastened with adhesive plaster. It has been found that shortening, angulation or over-riding of the fractured fragments can be corrected, and the correction can be maintained, after reduction by this method, and that free use of the entire hand and wrist is allowed without the hindrance of casts or splints. If extreme care is taken to ensure asepsis, the wires may be inserted in the surgery, "Novocain" being used. The authors describe their method in detail. They conclude by pointing out that internal fixation maintains proper reduction without slipping of the fragments, and that patients are grateful for the full use of their hands.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on October 13, 1943, at the Children's Hospital, Carlton, Melbourne, DR. HOWARD BOYD GRAHAM, the acting President, in the chair.

Influenzal Meningitis.

DR. A. G. NICHOLSON read a paper entitled "Meningitis due to Hæmophilus Influenzæ: Review of Treatment" (see page 320).

DR. H. MCLORINAN said that during the last fifteen months twelve patients suffering from influenzal meningitis had been admitted to the Queen's Memorial Infectious Diseases Hospital at Fairfield. In the previous twelve months only two patients were admitted. There had therefore been a striking increase in the last year. In one month six patients were admitted; but one could not regard this incidence as an epidemic. There had been seven deaths among twelve patients, one dying within two hours of admission to hospital. Treatment was found to be unsatisfactory, but it appeared that some response was obtained to sulphapyridine. Dr. McLorinan said that he had not read the article in the *Journal of Pediatrics* to which Dr. Nicholson had referred, but he had heard that the Commonwealth Serum Laboratories had elaborated a serum. He had telephoned Dr. Morgan, but did not find him enthusiastic about the results. The serum was tried in only one case at Fairfield. The death rate was eight cases out of fourteen. Topley and Wilson's figures in 1936 gave a mortality rate of 95%. Dr. McLorinan said he thought there had been some response to the sulphonamide drugs, especially to sulphapyridine. Sulphadiazine was used in two cases. Influenzal meningitis seemed to be a disease of childhood. The youngest patient was aged six months and the eldest twelve years. The older the child, the greater the chance of recovery. The dosage of sulphonamide compounds was large, but varied. The girl of twelve years was given 165 grammes of sulphapyridine in twenty-eight days. A boy, aged five years, was given 224 grammes of sulphapyridine and sulphathiazole over a period of ten weeks; this patient needed "Pentnucleotide" for the agranulocytosis which developed. In the case in which the serum was given, Dr. McLorinan said he thought that the child might have recovered in any case, so that he was unable to add much to Dr. Nicholson's work. Thirty cubic centimetres of serum were given intravenously and thirty cubic centimetres intramuscularly after sulphapyridine had been given for ten days. In this case relapse had occurred when administration of the drug was stopped. Relapse after stoppage of the drug was not an infrequent finding. Dr. McLorinan said he believed there was a case for the sulphonamide group of drugs in influenzal meningitis, but he was not enamoured of serum treatment. He had been disappointed with all antibacterial serums. Probably many of them were polyvalent, and so not specific. This was so in pneumococcal infection, and treatment with such serum was disappointing, as it was in meningococcal infection. In conclusion, Dr. McLorinan expressed the hope that the solution of the problem of treatment in these cases of influenzal meningitis would be found in one of the sulphonamide group of drugs, possibly one of the later additions, or perhaps it might be found amongst the penicillin group, which were capable of intrathecal administration.

DR. MONA BLANCH said she had recently lost a patient, aged one year, from influenzal meningitis, in spite of prolonged and expensive sulphanilamide therapy. The child was first examined one night at 9 p.m. There was a history of vomiting and limpness. The temperature was 103° F. and the child was pale. There was a suspicion of neck stiffness. The diagnosis appeared to lie between pyelitis and meningitis. The next morning the urine was examined, and this proved to be clear. Slight neck stiffness was present. Lumbar puncture was performed, and the cerebrospinal fluid was found to contain 5,360 polymorphonuclear leucocytes per cubic millimetre, and a few Gram-negative bacilli. Treatment was commenced with two grammes of "M & B 693" and was maintained with one gramme given every four hours. During the next fourteen days, 58 grammes were administered. The temperature fell, but rose again. A second lumbar puncture was performed, and the cerebro-spinal fluid contained 1,000 polymorphonuclear leucocytes per cubic millimetre. The fluid obtained at a third lumbar puncture after two weeks yielded influenzal bacilli, in spite of the extensive treatment. At this stage a change was made to sulphathiazole, but without much improvement. The child was then given saline and glucose solution by the intravenous drip method, soluble "M & B 693" being included. The temperature dropped for twenty-four hours, but the patient died soon afterwards, after having received a total of 134 grammes of sulphanilamide in thirty-four days. At the autopsy, masses of intensely greenish pus were found over the base of the brain, but very little over the cortex. Dr. Blanch said there were a few points in the case which should be made clear. Even after massive dosage the blood sulphanilamide content was found to be only four grammes per 100 cubic centimetres. This was one reason for the intravenous introduction of "M & B 693". The child was vomiting, but did not appear to vomit the tablets; the dose was repeated if this was thought to have occurred. Two blood transfusions were given to the patient, the first on her admission to hospital and the other seven days before she died. Finally it was noticed at the cerebrospinal fluid examination that the number of polymorphonuclear leucocytes was dropping, but the number of bacilli was increasing.

DR. J. W. GRIEVE said that in the days before the introduction of sulphanilamide there had been only one recovery at the Children's Hospital in children aged under twelve years. With sulphanilamide treatment one was encouraged at first by the results obtained; but the more recent results had not been so promising. Children suffering from this condition previously died within three weeks. The illness had been prolonged by sulphonamide treatment. There was, however, a greater percentage of true recoveries. With regard to the onset, Dr. Grieve said he agreed that neck stiffness was an important sign. There was something about such cases which made one think of meningitis. A history of vomiting, irritability and drowsiness was especially important. Urinary infection had to be eliminated. Dr. Grieve said he had noticed an increase in the incidence of the disease. The illness was characterized by remissions and relapse. He had noticed also that the older patients progressed more satisfactorily. This was noticeable before the introduction of sulphanilamide. Treatment with serum was under review. The number of cases was too small for dogmatic conclusions to be drawn. Dr. Grieve said he looked forward with interest to what the future held for special drug therapy and serum treatment in these cases, and Dr. Nicholson was to be congratulated on his paper.

DR. ROBERT SOUTHBY said that he recalled, as a resident medical officer, experiencing difficulty in performing lumbar puncture in such cases. He wondered whether sulphanilamide would have any effect towards thinning out the exudate. Frequently he found it was necessary to resort to cisternal puncture, and he wondered whether Dr. Nicholson had had to do the same. Dr. Southby said he was also interested in the tolerance displayed to the drug. Babies appeared to thrive on the drugs, and he had seen a fairly strong adult become anæmic and uræmic on five grammes per day. Dr. Southby asked Dr. Nicholson whether he could tell him the maximum dosage possible.

DR. LAWRENCE STOKES said that he had noticed in other cases the point made by Dr. Blanch concerning the relatively low concentration of sulphonamides in the blood, in spite of enormous dosage. Sometimes high initial figures might be obtained; but after a relapse, further "pushing" of the drug did not appear to elevate the blood concentration. Dr. Stokes said he was unable to explain this anomaly.

DR. HOWARD BOYD GRAHAM asked Dr. Nicholson whether he would, in his reply, refer briefly to the terminology in the disease.

Dr. Nicholson, in reply, thanked the speakers for their remarks. In answer to Dr. McLorinan, he said that spontaneous recovery was possible, and this made it difficult to assess the results of treatment. The sulphonamides had lowered the mortality figures in influenzal meningitis. These drugs *per se* had no effect on the organisms, and it was the response of the body to the organisms that effected the cure. Any addition to the body's defences should materially assist recovery. Recent reports on the use of sulphadiazine promised better results. Alexander used sulphanilamide and serum in comparatively small doses and obtained a reduction in the mortality rate to 28%. Dr. Nicholson said that in the successful cases he had been impressed by the clinical condition of the children. In those cases in which treatment by sulphanilamide alone had been successful, the course of the disease had been "rocky"; but this was not so in the case of the two children to whom serum had also been administered. Moreover, the course of the illness had been

shortened from eight or ten weeks to two or three weeks. In reply to Dr. Blanch, Dr. Nicholson said he had noticed the peculiar feature of the cerebro-spinal fluid yielding growth of organisms after long periods of therapy. The reason was obscure. It might be due to pockets in the cerebro-spinal spaces harbouring the organisms, which became active when the administration of the drug had been stopped. In answer to Dr. Southby, Dr. Nicholson said that he had experienced no difficulty in performing lumbar puncture in these cases. He had struck trouble in other cases, notably in meningococcal meningitis. He recalled one child from whom no fluid was obtained by the lumbar route on four occasions. However, after treatment no difficulty was experienced, and there was a free flow of cerebro-spinal fluid. It was a difficult matter to assess the maximum dosage. Different people required different dosage. The older the child, the more intolerant he became to the drug. This was noticeable both with meningococcal and with influenzal infection. Dr. Nicholson said that his experiences were similar to those of Dr. Stokes. He was unable to obtain the high concentration in the blood that might be expected. The highest concentration obtained was 4·2 milligrammes per 100 cubic centimetres in a child who had been given over 395 grammes. This observation did not appear to hold in pneumococcal infections. The terminology frequently used in this disease was wrong. These infections should not be called influenzal meningitis, any more than the common cold should be called influenza. There was no relationship between the influenza virus and the *Hæmophilus influenzæ* or Pfeiffer's bacillus as it was sometimes called, except that the two might sometimes be associated in the respiratory tract. In conclusion, Dr. Nicholson said that treatment with serum and sulphanilamide had had its "ups and downs", and the figures he presented were indeed too few for scientific deductions to be made. However, he was impressed with the good clinical progress and the short duration of the illness in his cases. If these were considered along with Alexander's figures, it would appear that the outlook had been improved.

Naval, Military and Air Force.

CLINICAL MEETING AT AN AUSTRALIAN GENERAL HOSPITAL.

A CLINICAL MEETING was held at an Australian general hospital on December 19, 1943. COLONEL KIRKWOOD, the Commanding Officer, presided and welcomed guests of the allied forces and local medical practitioners. Part of this report was published in the issues of March 18, 1944, and April 1, 1944.

Diseases of the Lungs.

MAJOR GERALD C. MOSS showed five patients suffering from diseases of the lungs. The first patient was aged forty-nine years and was considered to have a carcinoma of the lung probably originating in the left upper lobe bronchus. A non-productive cough had been present for two months. When he was first examined in hospital his temperature was normal, he had little sputum, his condition was good and there were no physical signs. Shortly after his admission to hospital an irregular pyrexia began, and it had persisted. The amount of sputum had steadily increased to twelve or fifteen ounces in twenty-four hours. It had been repeatedly examined for tubercle bacilli with negative results; various organisms were present, but no cells resembling carcinoma cells were seen. Clubbing of the fingers had occurred since his admission to hospital. Increasing dulness of the upper zone of the left lung had developed, and the breath sounds had become more tubular in character. X-ray examination revealed an infiltration in the left upper lobe, and serial X-ray films revealed its extension and increased density. No abscess cavity could be detected. A bronchoscopic examination was made by Major Blomfield; purulent material could be seen welling from the left main bronchus, but no other abnormality was found. The patient was not considered well enough for the subsequent instillation of lipiodol. The view taken was that gross infection (pneumonitis) was present, but that the clinical features strongly suggested that it was secondary to a bronchial carcinoma. Treatment, which had had little effect, had consisted of a course of sulphathiazole and postural drainage in the appropriate position.

Major Moss's second and third patients were suffering from bronchiectasis. In the case of one soldier, aged twenty years, a bronchogram revealed gross cylindrical dilatation confined to the left lower lobe. He had had a number of small hæmoptyses, and the daily amount of sputum was two or three ounces. It was not offensive. When everything was taken into consideration, it was thought that lobectomy would be justified. His present condition was good, but was likely to grow worse, and the bronchiectasis was likely to extend in the course of years.

In the case of another soldier, aged twenty-two years, both the upper and lower lobes of the left lung were affected. He was producing several ounces of offensive sputum per day. His general condition was also good. It was pointed out that extirpation of the whole lung would be necessary to cure him, and that the risk would be greater than in the other case. It was nevertheless decided to refer both patients for the opinion of a thoracic surgeon.

Major Moss's fourth patient had had an acute spontaneous pneumothorax with complete collapse of the left lung on September 22, 1943. At the time of the meeting his lung had reexpanded. His condition was good, although he had anxiety symptoms. He had been treated for acute dry pleurisy of the left side eight and three years previously; it was mentioned that these illnesses could have been attacks of spontaneous pneumothorax. The reasons for thinking the attack to be of non-tuberculous origin were given; and reference was made to the belief, in rare cases substantiated by X-ray examination, that attacks in some cases were due to rupture of an emphysematous bulla.

Major Moss's last patient was suffering from symptomless pulmonary tuberculosis discovered by routine X-ray examination. The patient, aged twenty-three years, had suffered from left *otitis media* three years earlier. He had been admitted to a hospital with a mild recrudescence of discharge from the ear. A routine X-ray examination of the chest was made. The film revealed infiltration in both upper zones, the right side being more involved than the left. At no time had he had cough, sputum or any other symptoms. There was no family history of tuberculosis. Sputum being unobtainable, the gastric juice was examined, and on two occasions tubercle bacilli were recovered by the concentration method. He was apyrexial, and there was only a slight increase in the blood sedimentation rate.

Major Moss, in a short discussion, then emphasized some points in the differential diagnosis of some non-tuberculous diseases of the lung. The discussion opened with some brief remarks on symptoms, physical signs and sputum examination. A number of lantern slides of plain X-ray films and bronchograms was then shown. Bronchial carcinoma of various types was illustrated, the slides showing pleural effusion, massive collapse of various lobes, obstruction to lipiodol by bronchial carcinoma, infiltration, pneumonitis, raised diaphragm from phrenic nerve paralysis *et cetera*. Other slides were from cases of lung abscess, of bronchiectasis with and without collapse of a lobe, and associated with pneumonitis (bronchiectatic abscess), congenital cystic disease of the lung, collapse of a lobe from a foreign body (a peanut) in a lower lobe bronchus, and others.

Major Moss concluded by saying that, although modern accessory methods of investigation enabled a diagnosis to be made in a considerable percentage of cases, the clinician not infrequently had to rely on his careful clinical appraisal and his experience of similar cases. This was the position with the patient who had been shown, and whose pneumonitis was considered to be secondary to a bronchial carcinoma, although no direct evidence of a bronchial carcinoma was forthcoming.

Radiological Exhibit.

Major Moss also showed, with short clinical records, some interesting X-ray prints of patients who had left the hospital. Included amongst these was a series showing gross congestion in the lung fields in a patient, aged forty-nine years, with chronic alcoholism, *B* (especially B_1) avitaminosis, Korsakoff's syndrome, peripheral neuritis, and congestive heart failure of beriberi type. The patient had made a rapid recovery with massive doses of vitamin B_1 given parenterally, together with a diet rich in vitamins. Nicotinic acid in amounts available had also been given. Serial X-ray films showed the disappearance of the infiltration in the lung fields. During a control period of twelve days, in which digitalis and injections of "Mersalyl" had been given at a camp hospital, there had been no improvement except slight decrease in the œdema of the legs. Another patient, aged fifty-one years, had received shrapnel

wounds in the left side of the chest in 1918. He had undergone several operations. He had been well until nine years prior to the meeting, when there was some discharge of pus through the skin. An abscess was apparently incised. He then remained well until one week before his admission to hospital, when he had several small hæmoptyses. X-ray examination revealed a foreign body (shrapnel) in the mid-zone of the left lung. A bronchogram revealed, in addition, localized saccular bronchiectasis in the left subapical region.

Pathological Exhibit.

CAPTAIN H. B. PENFOLD and the laboratory staff demonstrated pathological specimens of current interest. Microscopic preparations were shown to illustrate the various stages and types of malarial parasites and some intestinal protozoa. The procedure adopted to isolate and identify dysentery organisms was outlined. The falling drop method of determining the specific gravity of whole blood and plasma was demonstrated, and it was pointed out that from these specific gravities the hæmoglobin value, hæmatocrit reading and plasma content could be readily and reasonably accurately ascertained. Such information was of assistance in deciding whether, and how much, whole blood, serum or crystalloid solutions should be administered intravenously in cases of burns and other conditions. Cultures and smears of two strains of *Clostridium welchii* isolated from a case of gas gangrene were shown.

(To be continued.)

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 53, of March 16, 1943.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

Flight Lieutenant H. E. Williams (255176) is granted the acting rank of Squadron Leader whilst occupying a Squadron Leader post with effect from 13th December, 1943.—(Ex. Min. No. 73—Approved 15th March, 1944.)

Arthur Liddon Webb, M.B., B.S., F.R.C.S. (267631) is appointed to a commission on probation with the rank of Flight Lieutenant for part-time duty with effect from 10th January, 1944, and is promoted to the temporary rank of Squadron Leader with effect from the same date.

Robert Fruchtmann, M.D. (Poland) (277480), is appointed to a commission on probation with the rank of Flight Lieutenant with effect from 16th January, 1944.

The following officers are transferred from the Medical Branch Reserve to the Active List for full-time duty with effect from 16th January, 1944: (Flight Lieutenants) J. I. Guenther (287414), A. J. Gumley (287424), A. D. Packer (287403), K. C. Porter (287404), R. T. Steele (287406), C. D. Swaine (287405).

The probationary appointments of the following Flight Lieutenants are confirmed: E. W. Gibson (266785), P. G. D. Prentice (277336), D. Gordon (276870), J. W. P. Henderson (277272), D. D. Letham (277337), P. A. Tod (277138), C. Roe (277273), N. B. Howse (267413), D. F. O'Brien (253180).

Reserve: Medical Branch.

The following temporary Squadron Leaders are transferred from the Active List at their own request with effect from the dates indicated: A. H. Penington (251211), 14th January, 1944, D. C. Trainor (261653), 25th January, 1944.—(Ex. Min. No. 71—Approved 15th March, 1944.)

Garry Edward Wilbur Bennett, M.B., B.S. (257613), is appointed to a commission on probation with the rank of Flight Lieutenant with effect from 26th January, 1944.—(Ex. Min. No. 75—Approved 15th March, 1944.)

Correspondence.

HOUSEKEEPERS' EMERGENCY SERVICE.

SIR: I have been asked to draw the attention of your readers to the existence of the Housekeepers' Emergency Service, as it is possible that medical practitioners might be glad to make use of it for their patients. Members of the medical profession are probably acutely aware of the difficulty experienced by women in making suitable arrangements for the care of their homes and children in times of sickness, childbirth or other emergency. In an attempt to meet this community need, which has been accentuated by the war, the Housekeepers' Emergency Service Committee was established on November 1, 1943, to provide temporary household help at such times.

Lady Wakehurst is patron of the committee, which consists of representatives of the National Council of Women, District Nursing Association, New South Wales Society for Crippled Children, Royal Society for the Welfare of Mothers and Babies, Red Cross Society, Country Women's Association, Australian Comforts Fund Family Welfare Bureau, the Domestic Employees' Union, and the Women's Auxiliary National Services.

The State Government has made a grant and generous contributions have been received from various organizations and people who are interested in social welfare. The families who are served by the housekeepers contribute according to their means; the salaries of the housekeepers are paid continuously by the committee.

The housekeepers are carefully selected for their sense of responsibility and practical ability, their love of children and real spirit of service.

The arrangements to provide this help for any family are made according to the medical recommendations. The housekeepers can stay in the homes in the mothers' absence or visit daily, according to the individual need. No definite limit has been set to the period for which a housekeeper may serve any one family, but the committee hopes that they may not often be required for longer than one month.

Inquiries can be made from Mrs. Fetherstonhaugh, Organizing Secretary, 34, Martin Place, BW 4495, extension 28.

Yours, etc.,

ALISON PLAYER,
Honorary Secretary, Housekeepers' Emergency Service.

34, Martin Place,
Sydney,
March 24, 1944.

HEALTH AND THE GOLD STANDARD.

SIR: To find a scientific journal of your standing denouncing the gold standard at the present time is encouraging. Can discussion of this vital problem by scientific men be stimulated? I believe that groundless fears of some financial experts can best be relieved by repeating the established fact that a paper pound in Australia could buy more of everything that mattered in 1939 than it could in 1929, although it could buy only half as much gold. Letting the price of gold rise 100% did no harm to our money. A gold standard simply means holding gold to a fixed price. Any price can be selected. But for governments to attach a fixed value in money to a commodity like gold is an absurdity, as Dr. Dale points out so clearly.

It was a pity, if I may say so, that a reckless attack on banking was mixed up with the clear argument. Fifteen years ago, when in the throes of the slump, there was much outcry against the credit structure. It was rightly felt that the depression was man made and due to distorted high finance, but the root of the trouble was not discovered for some time. Every country that recovered from the slump benefited by unpegging the price of gold and letting it rise naturally to a limited extent. The credit structure was not materially altered. It should be recognized that to every credit there is an equal and opposite debit. When Smith lends Robinson money, credit and debit are created. When repayment is made the *plus* and *minus* coalesce and both vanish. Trading banks have little power in the matter; in fact, the credit may be regarded as created when the depositor puts his money in the bank rather than when the bank lends it as part of an overdraft. Banks don't lend more than they get. We should not be dismayed because we have to grapple with a credit system so much larger than it was when the slump developed. The responsibility for currency rests with the governments or more particularly with their treasuries. It may be true that the British Treasury preferred to evade its responsibility by claiming that it acted on the advice of the Bank of England. In recent years other countries have been strengthening central banks to shelter their governments. The foolishness of fixing the price of gold was committed by governments in all countries, not by banks. The present-day foolishness

(before the war started) of imprisoning gold was the act of governments, not banks. That action is like caging the beast, whereas before it was hobbled. The constraint was set up by bureaucracy, not by the banking system. I do not think the great men of finance are malicious so much as befogged. Scientific argument can do much to throw light on their problem.

Some day, perhaps a few years after the war, gold may be allowed to trickle out from its prison and serve mankind in freedom once again. What form will it take? Historically the size of coin most favoured by man has always been that of the sovereign or guinea, which, by the way, was not a royal coin, but separate from the currency of the realm. Such gold coins will probably not be used for paying wages as was customary with the sovereign before 1914 because they may be expected to fluctuate in value from day to day as gold coins should. Such gold coins form an elastic link between the real world of commodities and the money system, whereas the gold standard imposed a rigid link. That rigidity, it is now thought, set up the damaging constraints which caused the slump. The modern monetary system did not have to be amended. After most careful scrutiny from within and without it has progressed along the same efficient lines as before the depression.

It seems clear that Australia should avoid all international conspiracies concerned with finance, foreign exchange, gold and stabilization. Your pages 237 to 242 should do much to lead thought in that direction.

Yours, etc.,

GEORGE F. DAVIDSON, O.B.E., M.A. (Cantab.), B.E. (Sydney), M.I.Mech.E. (London).

1, Springfield Avenue,
Potts Point,
Sydney.
March 25, 1944.

SIR: Dr. John Dale's article is most interesting and informative in its historical aspects, but his description of the "banking system" and his indictments of the "monetary system" as the cause of depression and poverty cannot be accepted as correct or fundamental.

To give a detailed and complete refutation of his ideas would require an article as long as his lecture. It has been done by others. However, I will mention just a few points.

In treating of the circulation of gold as money he states, "the sovereigns came back into the banks which were, therefore, as you were, *plus* the new gold, *plus* the ability to expand their loans". He fails to explain that as the gold tokens returned to the bank they were not owned by the bank and that they were only loaned by the bank. If any equivalent amount of wealth in fact more than equivalent amount of wealth or claims to wealth were deposited by the borrower, and further the gold tokens returning to the bank represented real wealth created, or services rendered.

He states that the issue of money determines the production of goods; now the exact reverse is the case, as he himself shows in his tracing the history of money. The mere issue of money does not determine the production of goods. The factors that determine the production of goods are human desire, and the facilities enabling human beings to obtain access to the raw materials of all wealth, that is, natural resources.

Further, if the issue of gold or money were the factor that called forth the production of goods, how is it that Spain and Portugal were not enriched by the advent of vast quantities of the precious metals from America, but rather the reverse. It was one of the many factors in their impoverishment, as he himself states, prices rose 600%. Can he seriously maintain that such a rise in prices would cause or did cause prosperity? It might enrich a few for a time, but it, of course, was a factor in widespread disaster. A rise in price only benefits those who hold natural resources. It impoverishes those who do not own them.

He apparently approves of the Marxian theory of value. This is one of the fundamentals of Marxian economics and has been proven erroneous; in fact, common observation shows that the labour incorporated in a commodity has nothing necessarily to do with its value. A "silk hat" for instance might have had considerable "labour value" incorporated in it, but you could get nothing for it today. I will sell one for a shilling, but it is not worth even that, for no one desires it. Again, he states that the war itself with the immensely increased production required has necessitated a great increase in the supply of money. Is not this statement seriously near a contradiction of his previous assertion that it is money that calls for production? The war called for the production of more wealth and money was created by the Government to pay for this wealth; in reality it has to be paid for by future production of wealth. I am in full agreement with Dr. Dale in his enthusiasm for liberty and democracy, but it is an unwarrantable assumption to assert that it is our monetary system that is the keystone of these desiderals. It may be faulty. No human system is perfect, but I would say without fear of contradiction that the great danger to our liberty comes from the conferring of privilege to some at the expense of others. Can Dr. Dale point to any greater form of privilege or more potent for evil than the private ownership of the earth?

If I owned all Australia and he owned all the money in Australia, with whom would the power reside?

His money would be powerless to oppress me, but my ownership of the land would give me the power over his very life itself.

Yours, etc.,

PAUL G. DANE.

111, Collins Street,
Melbourne, C.1,
March 28, 1944.

ACUTE OTITIS MEDIA—PARACENTESIS—SULPHONAMIDE DRUGS.

SIR: Discussions have taken place periodically about the desirability of early paracentesis in cases of acute *otitis media*. Without doubt it is a powerful aid in the prevention of deafness by minimizing the damage to the mucosa of the middle ear and consequent scarring. During the last few years sulphonamide drugs have come into common use; and when given to patients suffering from *otitis media* the inflammation may settle down without either paracentesis being performed or spontaneous rupture occurring, with consequent discharge.

Each of the undersigned has seen a few cases that had been so treated, and the result had been most unfortunate because the exudate in the middle ear had become organized, ending in a chronic adhesive process of the middle ear. This is a quite intractable condition accompanied by considerable and progressive deafness.

The same thing has been noted in England, and a report appeared in a letter in the *British Medical Journal* of November 20, 1943. We think it is important enough to ask you to publish this letter to warn Australian practitioners how necessary it is to avoid deafness after otitis as well as to cure the otitis. When there is exudate in the middle ear and the drum is bulging, paracentesis should be done to evacuate the exudate, notwithstanding that adequate chemotherapy is administered. Care should be taken to examine the hearing also, to make sure that there is no residual deafness. If recovery of hearing is delayed, timely air inflation of the middle ear may prove very valuable.

Yours, etc.,

ERIC P. BLASHKI.
GUSTAV BONDY.

193, Macquarie Street,
Sydney,
March 28, 1944.

INDUSTRIAL MEDICINE.

SIR: It was with great interest that I read Dr. Nelson's paper on industrial medicine in your issue of March 25 last. The comments of Dr. Stormon, Dr. Finlayson and Dr. Kesteven also contain much constructive comment on the side which interests me most, namely, industrial dermatitis.

I think I am the person referred to by Dr. Stormon as denying the existence of oil dermatitis. Especially since the present war began I have been compelled to take a larger share in industrial dermatology, and I have never yet seen a *dermatitis eczematosa* due to lubricating oil, cutting compound, soluble oil or grease, in spite of having examined scores of patients said to be suffering from oil dermatitis.

In some instances dermatitis does occur in machine workers, but in every case seen by me the method of cleaning up after work or some outside influence has been shown responsible. The enthusiastic first-aid man is the

worst offender. Such men think antiseptics can do no harm, and one caused an epidemic of dermatitis of external irritant origin because, having found one proprietery antiseptic in pure form a good means of removing grease and dirt, he recommended it to the workers for that purpose.

Again, if unsuitable soaps with free caustic soda or potash do set up a mild dermatitis, the first-aid man frequently makes matters serious by applying strong antiseptic applications under the belief that dermatitis is due to an infection. It should be impressed on all nurses and first-aid men that occupational dermatitis is due to chemical irritant action and not to bacterial infection, and that all antiseptics are capable of irritant action on some skins, especially those already in rebellion against chemical irritant effect from other sources.

To return to the "oil dots" which masquerade under the title of "oil dermatitis" or "oil folliculitis", it is obvious that this condition bears no relation to eczema and very rarely shows any inflammation (only when foreign body action has enabled suppuration to occur round the plug of oil or dirt). It really is not even a folliculitis because there is no inflammation to warrant the "itis".

It causes no disability except to a trifling extent when an odd follicle suppurates. The lesions invariably consist of small black plugs of metal dust, dirt and oil which have been rubbed into the lanugo hair follicles or sebaceous follicles of the skin. It is the oil soaked and dirty working clothes that are most frequently responsible. Plenty of hot water and ordinary soap to skin and to clothes will prevent it, and as Dr. Kesteven mentioned, pinching massage helps to clear it. But it is not a true dermatitis (eczematosa) and it not due to chemical irritant action.

Finally, I want to point out that the use of the word "dermatitis", unless qualified by "seborrhœic", "herpetiformis", "artefacta" or the like, means an eczematous outbreak (almost always of external irritant action of chemical nature). It is confusing, especially in court, to find the name applied as a generic term for any old inflammation of the skin. It should never be used to designate any disease of the skin due to infection, at least without a qualifying adjective, such as "seborrhœic".

Yours, etc.,

E. H. MOLESWORTH.

Beanbah,
235, Macquarie Street,
Sydney.
March 29, 1944.

Obituary.

HORACE FREDERICK HAYES.

WE regret to announce the death of Dr. Horace Frederick Hayes, which occurred on March 26, 1944, at Caulfield, Victoria.

ROY WILLIAM CHAMBERS.

WE regret to announce the death of Dr. Roy William Chambers, which occurred on March 28, 1944, at Melbourne.

HERCULES BRADSHAW MOORHEAD.

WE regret to announce the death of Dr. Hercules Bradshaw Moorhead, which occurred on March 29, 1944, at Hobart.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned has been registered as a duly qualified medical practitioner:

Gollan, John Alexander, M.B., B.S., 1931 (Univ. Melbourne), Hobart.

Medical Appointments.

Sir Raphael West Cilento, Dr. Valentine McDowall, Dr. Aubrey David Dick Pye and Dr. Henry Joseph Windsor have been appointed members of the Queensland Radium Institute.

Dr. Walter Ernest Summons and Dr. Frank Victor Gordon Scholes have been appointed members of the Commission of Public Health, Victoria.

Diary for the Month.

APR. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 11.—New South Wales Branch, B.M.A.: Organization and Science Committee.
APR. 11.—Tasmanian Branch, B.M.A.: Branch Meeting.
APR. 14.—Queensland Branch, B.M.A.: Council Meeting.
APR. 14.—Victorian Branch, B.M.A.: Ethics Subcommittee.
APR. 17.—Victorian Branch, B.M.A.: Hospital Subcommittee.
APR. 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
APR. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 18.—Victorian Branch, B.M.A.: Organization Subcommittee.
APR. 19.—Western Australian Branch, B.M.A.: Branch Meeting.
APR. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
APR. 20.—Victorian Branch, B.M.A.: Executive Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be Stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.